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SILICOSIS ISSUE

The Statistical Characteristics of Dust Phthisis (Pulmonary Silicosis)

The Occurrence and Clinical Manifestations of Silicosis among Hard Ground Workers in Coal Mines

Exposure to Silica Dust without the Occurrence of Silicosis

Experimental Silicosis

A Short Description of a Dust Trap to Be Used in Conjunction with Percussive Rock Drills

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THE STATISTICAL CHARACTERISTICS OF DUST PHTHISIS (PULMONARY SILICOSIS)*

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PULMONARY silicosis is now a well-recognized clinical and pathologic entity. It also possesses a statistical entity at least as characteristic as that of any cause of death known to epidemiologists.

The condition is brought about by exposure for a series of years to the inhalation of fine particles of silica dust; the finer the particles and the more intensive the exposure, the more rapidly does the condition develop. The majority of the particles are little more than 1 micron in size and so are sufficiently small to penetrate the bronchioles and gain access to the alveoli.

No condition comparable with silicosis has been described as following on the inhalation of any other dust. Nor has such inhalation been found associated with the characteristic mortality data to be presently described.

Simple silicosis, although it may end fatally without any intervening complication, does not readily incapacitate those affected. In time, however, as more and more of the pulmonary tissue is thrown out of action, shortness of breath on exertion becomes manifest, a persistent cough develops, and strain is thrown on the heart's action owing to resistance in the pulmonary circulation. Deaths in such cases may be attributed to pulmonary fibrosis.

As the pulmonary apparatus becomes more and more damaged, it tends more and more to succumb to such respiratory diseases as bronchitis, pneumonia, and tuberculosis.

The dust of other materials acts differently from silica dust and each

* Received for publication June 7, 1926.

type of dust has its own peculiarity. It may be soluble in the body fluids, as limestone is, and cause no trouble. Or, although insoluble, the dust particles may seldom be below 5 microns in size, and so, while entering the bronchi, may not be able to penetrate into the finer bronchioles and alveoli; such dust may be expected to originate bronchitis without leading to pneumonia. Other dusts, particularly dusts of insoluble inorganic material, such as emery, are sufficiently small to affect the air passages from the bronchi to the alveoli; they increase the liability to contract bronchitis and pneumonia.

Data from Mortality Records, 1921 to 1923

The statements which are here being made were deduced from existing mortality records which have been stated elsewhere; but in this communication, only the most recent data will be quoted in support—the data of occupational mortality in England and Wales for 1921 to 1923.[1] The grouping of these data has been specially arranged so as to put the statements previously made to the acid test of prophecy. They are found to stand the test well, even though the smallness of the populations at risk makes some of the mortality rates uncertain and unreliable; the general trend rather than the absolute magnitude of the figures should be observed.

Bronchitis

Of the occupations instanced, all those with a silica dust risk experience a high mortality from bronchitis (see Table 1). In contrast, the limestone quarrier does not. The limestone mason is probably not a pure occupational group, since the grouping refers to masons living at the date of the census, and dying during 1921 to 1923, in counties where limestone or sandstone is nearly exclusively worked; but masons during their life tend to work on different stones, and do not always remain living in the same district. The contrast to be made here is that between the two groups of masons; then the excess of bronchitis among the sandstone masons stands out clearly. Both the brick groups as well as glazers and polishers, when compared with the group for all males, suffer unduly from bronchitis.

Pneumonia

The silica risk occupations again show high mortality rates (see Table 2). Again the limestone quarrier exhibits no definite excess, while some slight excess for the limestone mason must, for reasons just given, be discounted. The brick groups exhibit no definite excess; possibly the dust particles are not small enough to reach the alveoli. The glazers and polishers exhibit definite excess; the dust to which they are exposed is varied, but includes particles of emery and oxide of aluminium small enough to reach the alveoli.

Phthisis

Silica dust alone possesses the further characteristic that it increases the liability to succumb also to pul-

[1] The author desires to express his appreciation of the courtesy extended to him by the Registrar-General in permitting access to these records before publication.

TABLE 1.—MORTALITY FROM BRONCHITIS AT CERTAIN AGE PERIODS PER THOUSAND LIVING: ENGLAND AND WALES, 1921 TO 1923

GROUP[1]	AGE PERIODS		
	35–	45–	55–64
All occupied and retired males	0.20	0.56	1.91
Tin, copper, and lead miners—underground workers (not superintending staff)[2]	0.56	2.66	9.04
Stone miners and quarriers: igneous	....	....	....
Stone miners and quarriers: limestone	0.15	0.35	1.86
Stone miners and quarriers: sandstone[2]	....	0.35	6.69
Masons, stone cutters and dressers: limestone	....	0.56	3.11
Masons, stone cutters and dressers: sandstone[2]	0.78	1.17	12.42
Brick and plain tile makers, molders, etc.; furnace and crucible pot makers	0.33	0.88	4.59
Brick, tile, etc., kiln and oven men	0.23	0.57	3.13
Potters' mill workers; slip makers; potters[2]	0.61	2.05	13.27
Earthenware, china, etc., kiln and oven men, setters and placers[2]	0.71	3.09	10.01
Cutlery trade: grinders[2]	1.15	3.68	16.15
Cutlery trade: glazers, polishers	0.26	0.74	4.57

[1] All the groups, except "occupied and retired males," have an occupational exposure to dust.

[2] Exposure to silica dust is present only in these occupations.

TABLE 2.—MORTALITY FROM PNEUMONIA AT CERTAIN AGE PERIODS PER THOUSAND LIVING: ENGLAND AND WALES, 1921 TO 1923

GROUP[1]	AGE PERIODS			
	25–	35–	45–	55–64
All occupied and retired males	0.40	0.72	1.03	1.70
Tin, copper, and lead miners—underground workers (not superintending staff)[2]	....	....	2.66	....
Stone miners and quarriers: igneous	0.48	0.45	....	....
Stone miners and quarriers: limestone	0.65	0.59	1.41	0.80
Stone miners and quarriers: sandstone[2]	1.61	1.86	1.73	3.82
Masons, stone cutters and dressers: limestone	....	0.48	1.48	2.34
Masons, stone cutters and dressers: sandstone[2]	0.67	0.65	2.34	1.84
Brick and plain tile makers, molders, etc.; furnace and crucible pot makers	0.48	0.50	1.11	0.77
Brick, tile, etc., kiln and oven men	....	0.46	0.86	2.09
Potters' mill workers; slip makers; potters[2]	0.34	1.21	1.10	2.47
Earthenware, china, etc., kiln and oven men, setters and placers[2]	0.25	1.88	1.69	2.86
Cutlery trade: grinders[2]	0.99	0.57	4.90	2.31
Cutlery trade: glazers, polishers	0.26	1.83	2.77	2.54

[1] All the groups, except "occupied and retired males," have an occupational exposure to dust.

[2] Exposure to silica dust is present only in these occupations.

monary tuberculosis. Why it possesses this curious characteristic is not at present fully understood, but there is fair agreement that its action is a chemical one due to the conversion of the fine dust particles in the lungs into colloidal silica, which acts as a definite tissue poison. This characteristic of silicosis is of immense statistical importance, since it leads to "phthisis" being returned as the cause of death for an unusual number of deaths among those employed in occupations with a silica risk. Some of these deaths may be due to simple silicosis—fibroid phthisis—but many more are due to tuberculosis superimposed upon silicosis. Statistically, when examining deaths among those exposed during life to a silica risk, all deaths from phthisis, whether fibroid or tuberculous, should be grouped together and compared with similar deaths in other occupational groups.

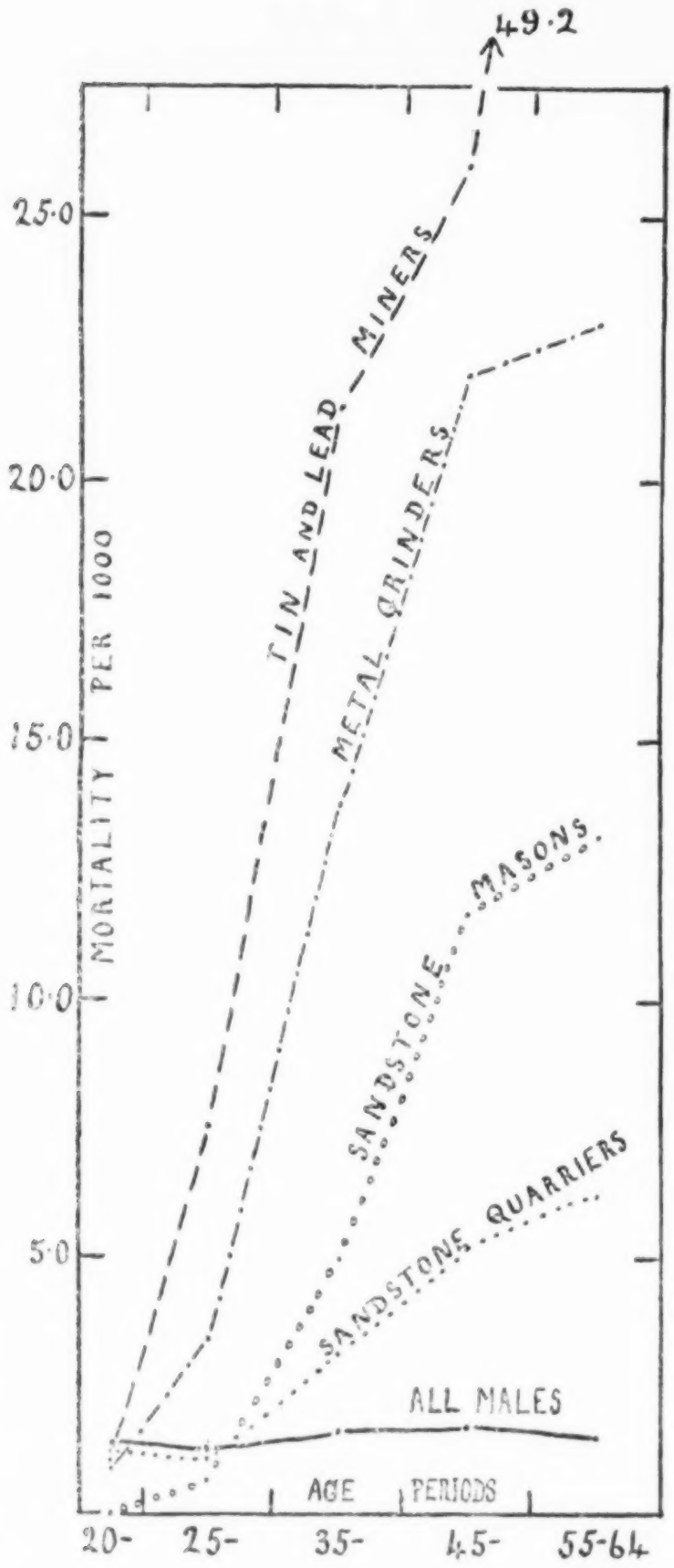

FIG. 1.—Mortality from pulmonary tuberculosis plus fibroid phthisis at certain age periods per thousand living: England and Wales, 1921 to 1923.

The new records, examined on this basis, bring out the evils of exposure to silica dust quite definitely (see Fig. 1); and moreover exhibit mortality rates closely correlated to the intensity of exposure to dust. The sandstone quarrier works much more in the open air than does the mason, who in his turn is less exposed to a confined dust charged atmosphere than is the metal grinder; while the metalliferous miner employed in underground workings is most exposed of all to the risk, and he experiences a corresponding mortality.

The contrast with the other dust occupations is sharp. The limestone mason shows some excess (see Table 3), which again must be discounted, and is slight compared with the excess of the sandstone mason. Glazers and polishers also exhibit some excess of phthisis mortality, which, however, is highest at the ages 35 to 44, an unusual thing for dust phthisis; possibly this excess, like that of printers and tailors, is due to increased exposure to infection rather than to dust risk.

Other occupations, having no dust risk, such as printers, shoemakers, and tailors, may and often do experience a high mortality from phthisis. Hence the mere existence of a high phthisis death rate is not distinctive of silicosis; but other associated causes of death assist in distinguishing these occupations from those with a silica risk.

The influence exerted by silica dust upon the air passages—an influence which predisposes to pneumonia and particularly to bronchitis—comes in to help. This influence causes in a "silica" group high mortalities from these diseases, which are conspicuously absent where there is no dust risk. When the phthisis mortality of a "silica" dust group, with its attendant high incidence of bronchitis and pneumonia, is compared with the phthisis mortality of a non-dust group, a further characteristic is found; *viz.*, the mortality rate for phthisis in the silica group always exhibits its highest incidence later in life, between the ages 55 and 64, than does ordinary pulmonary tuberculosis.

TABLE 3.—MORTALITY FROM PULMONARY TUBERCULOSIS PLUS FIBROID PHTHISIS AT CERTAIN AGE PERIODS PER THOUSAND LIVING: ENGLAND AND WALES, 1921 TO 1923

GROUP[1]		AGE PERIODS				
		20–	25–	35–	45–	55–64
All occupied and retired males		1.36	1.33	1.60	1.68	1.54
Tin, copper, and lead miners—underground workers (not superintending staff)[2]		1.00	7.52	21.43	25.95	49.20
Stone miners and quarriers	igneous	0.93	.....	0.90	0.50	2.93
	limestone	1.83	0.65	1.33	1.93	2.66
	sandstone[2]	1.22	1.08	3.10	5.19	6.21
Masons, stone cutters and dressers	limestone	0.97	1.50	3.23	5.56	2.53
	sandstone[2]	....	0.67	4.94	11.67	13.11
Brick and plain tile makers, molders, etc.; furnace and crucible pot makers		0.31	1.29	1.49	2.65	1.91
Brick, tile, etc., kiln and oven men		1.53	0.29	1.15	0.57	3.65
Potters' mill workers; slip makers; potters[2]		3.08	1.72	4.25	6.32	7.10
Earthenware, china, etc., kiln and oven men, setters and placers[2]		2.29	1.24	4.48	4.50	5.01
Cutlery trade	grinders[2]	0.93	3.46	13.77	22.06	23.17
	glazers, polishers	2.31	2.33	4.04	3.69	3.56

[1] All the groups, except "occupied and retired males," have an occupational exposure to dust.

[2] Exposure to silica dust is present only in these occupations.

The Coal Miner

This characteristic, which has been established from mortality records of definite silica groups, is important, quite apart from any light it may shed on the way in which silica acts. Thus, coal miners have always had a low death rate from phthisis, but this low death rate, since 1890 when statistical records first became available, has consistently shown a maximum inci-

dence late in life, between the ages 55 and 64, at the very period picked out by silicosis. Further, on the

TABLE 4.—MORTALITY AMONG SOME COAL MINERS AT CERTAIN AGE PERIODS PER THOUSAND LIVING: ENGLAND AND WALES, 1921 TO 1923

GROUP		AGE PERIODS				
		20–	25–	35–	45–	55–64
Bronchitis						
Hewers and getters	Lancashire	0.06	0.09	0.20	0.72	4.83
	Glamorgan	0.02	0.03	0.16	0.83	5.08
	Derbyshire	....	0.07	0.13	0.64	2.95
Others underground	Lancashire	0.04	0.17	0.28	0.99	4.68
	Glamorgan	....	0.03	0.42	1.00	4.63
	Derbyshire	....	0.24	....	1.01	0.89
All occupied and retired males		0.03	0.06	0.20	0.56	1.91
Pneumonia						
Hewers and getters	Lancashire	0.64	0.57	0.66	1.54	1.95
	Glamorgan	0.39	0.33	0.55	0.97	1.60
	Derbyshire	0.11	0.23	0.45	0.70	1.55
Others underground	Lancashire	0.47	0.30	0.89	2.13	2.73
	Glamorgan	0.46	0.45	0.67	1.22	3.16
	Derbyshire	....	0.73	1.36	1.01	1.33
All occupied and retired males		0.28	0.40	0.72	1.03	1.70
Pulmonary Tuberculosis plus Fibroid Phthisis						
Hewers and getters	Lancashire	1.33	0.86	1.88	1.60	2.23
	Glamorgan	0.84	0.65	1.06	1.31	1.78
	Derbyshire	0.17	0.72	0.58	0.89	0.70
Others underground	Lancashire	1.30	0.73	1.31	1.44	2.05
	Glamorgan	1.18	1.03	1.62	1.61	1.47
	Derbyshire	1.06	1.45	1.36	1.35	0.89
All occupied and retired males		1.36	1.33	1.61	1.68	1.54

different coal fields, the phthisis mortality, low as it is, has varied greatly, from a comparative figure of 53 in 1910 to 1912 on the Nottingham field to one of 107 on the Lancashire field. At the same time mortality from bronchitis has varied in a similar way from a figure of 40 for Nottingham to one of 100 for Lancashire; while the record for pneumonia tells the same story, and has varied from a figure of 25 for Nottingham to one of 88 for Lancashire. These high mortalities from bronchitis and pneumonia in Lancashire may be influenced by local climatic conditions; but, as the miners suffer more than the rest of the population, some occupational influence must also be concerned. There has been then among Lancashire miners (with South Wales miners not far behind) a phthisis mortality high for coal miners, with its greatest incidence late in life, and, associated with it, all the statistical characteristics so far men-

tioned of tuberculous silicosis are present. The position suggested[2] is that a certain amount of silicosis has long been present among some groups of coal miners, an amount insufficient to raise seriously the phthisis death rate for the occupation as a whole and only to be detected statistically by close attention to available mortality records. Its existence was indeed detected statistically before it was described clinically; and only now is evidence coming to light[3] that the disease is occurring among coal miners, but only among a small group employed in driving headings and in doing similar work in silica rocks.

TABLE 5.—MORTALITY FROM CHRONIC NEPHRITIS AT CERTAIN AGE PERIODS PER THOUSAND LIVING: ENGLAND AND WALES, 1921 TO 1923

GROUP[1]	AGE PERIODS	
	45–	55–64
All occupied and retired males	0.45	1.08
Tin, copper, and lead miners—underground workers (not superintending staff)[2]	3.33	2.01
Stone miners and quarriers: igneous	....	0.73
Stone miners and quarriers: limestone	0.18	0.27
Stone miners and quarriers: sandstone[2]	....	1.91
Masons, stone cutters and dressers: limestone	1.11	0.78
Masons, stone cutters and dressers: sandstone[2]	0.83	1.84
Brick and plain tile makers, molders, etc.; furnace and crucible pot makers	0.22	0.77
Brick, tile, etc., kiln and oven men	0.86	1.04
Potters' mill workers; slip makers; potters[2]	0.79	1.54
Earthenware, china, etc., kiln and oven men, setters and placers[2]	0.56	2.86
Cutlery trade: grinders[2]	1.23	1.15
Cutlery trade: glazers, polishers	0.74	1.02

[1] All the groups, except "occupied and retired males," have an occupational exposure to dust.

[2] Exposure to silica dust is present only in these occupations.

Here again the new records (see Table 4) bear out those of former years. The Lancashire miner, whether at the coal face or doing other work below ground, is still found heading the list, and suffering more from phthisis, bronchitis, and pneumonia than his fellow on other coal fields. The South Wales (Glamorgan) miner is not far behind. But the Derbyshire miner, as heretofore, provides a contrast with low mortalities for each of these respiratory diseases.

Chronic Nephritis

There is another characteristic of mortality figures referring to a silica group which, although not quite so definite as those already mentioned, is suggestive. Usually in any silica group the mortality from Bright's disease is

[2] Collis, E. L.: A Study of the Mortality of Coal Miners, England and Wales. THIS JOURN., 1922–1923, 4, 308–310.

[3] See article by Tattersall in this issue, p. 466.

high. The toxic action of silica in solution upon living tissues has been alluded to already; there is some evidence from animal experiments that silica is eliminated from the blood by the kidneys and that the kidneys are affected in the process. If we concede that silica dust carried into the lungs is slowly dissolved, the escape of some into the blood stream does not seem improbable. It might then be expected to act as it does in the animals, causing fibrosis of the kidneys as an end-result. Mortality from Bright's disease is also associated with overindulgence in alcohol, but, since mortality from cirrhosis of the liver and alcoholism is low rather than high in most silica groups, alcoholism cannot fairly be called in to explain the facts.

Here also the new records are of great interest (see Table 5). All the silica risk groups show some excess mortality from chronic nephritis, an excess which is highest where the excessive mortality from phthisis is highest. This excess is not a characteristic of the other occupational groups quoted.

Infectious Characteristics

One last statistical peculiarity may be mentioned, the certitude of which rests upon rather insecure data; but, when a large number of imperfect data all point in the same direction, the significance of the probable error attached to each set of records is much diminished, even though the extent is not open to methods of exact computation. The peculiarity is that those who come in contact with tuberculous silicotics, but who are not themselves silicotic, do not appear to contract tuberculosis, or at least they do not experience a phthisis mortality any higher than, if as high as, that of the ordinary population. The evidence on this point, although it has been published elsewhere, may be worth repeating on account of the number of observers who have independently reported this peculiarity. As long ago as the sixteenth century Agricola wrote of women in the Carpathian mountains who, owing to the high male mortality among the miners from this cause, married seven times. Thackrah similarly gives evidence that there were in the village of Arkendale (in the heart of the lead mining district) not less than thirty widows under 30 years of age. Greenhow, in 1858, said: "It is the injurious character of the male occupation which causes Alston, the most exclusively lead-mining district in England, to be the place in which there is a larger proportion of widows than in any other place in the kingdom." Peacock, in 1864, pointed out that among tin miners the disease "usually occurs in persons who do not present any hereditary predisposition to phthisis—their parents and other relatives having often attained advanced age and being quite healthy." In more recent years, as the result of investigations (1902 to 1904) into the nature and cause of excessive mortality among tin miners, Dr. Haldane observed that the wives and children of these men never seem to be affected, although occupying the same room as the affected men who never go to hospital but sit at home and expectorate sputum loaded with tubercle bacilli. In 1909 Steel-Maitland and Squire drew attention to the number of widows of tin miners in Cornwall.

and of ganister workers at Wortley, even though, due to the outnumbering of women by men in the latter district, the women often marry again. A paragraph in the daily press, which stated that there were in Brandon eleven widows of flint knappers, led to my inquiry, which established the same point for the flint knapping industry. Dr. Mathew Hay independently made the same observation in relation to female relatives of Aberdeen granite cutters. Dr. Wheatley has referred to the same point for Grinshill masons and their relatives. Dr. Barwise has noted that, although Derbyshire gritstone workers suffer in excess from phthisis, "the percentage of deaths of females living in stone-workers' houses is less than among agriculturists, although the proportion of quarrymen married is greater than that of the agricultural group." My own investigations into the mortality of stonemasons, their wives and widows, disclosed the same absence of any excessive phthisis mortality among the latter. Dr. Turner has told me that his inquiries in South Africa into the occurrence of phthisis in the native kraals have failed to establish that natives returning home with dust phthisis from gold mines disseminate the disease among the tribes. While Hoffman in 1922, after describing the extraordinary amount of phthisis among American granite cutters, writes of their wives that "the relative frequency of pulmonary tuberculosis among them is considerably less than that which would be theoretically assumed to occur."

Summary

1. Appeal to mortality records, and in particular to the most recent official statistics for England and Wales which have been specially prepared to bring out occupational risks, provides definite evidence that groups of workers exposed to a silica dust risk experience high mortalities from (*a*) bronchitis, (*b*) pneumonia, (*c*) phthisis, and (*d*) chronic nephritis.

This combination in the mortality records of any occupational group is statistically diagnostic of the presence of a silica dust risk.

2. Mortality from phthisis in an occupation with a silica dust risk always exhibits its highest incidence late in life.

3. Silicotic tuberculosis appears to be less infectious than ordinary tuberculosis.

THE OCCURRENCE AND CLINICAL MANIFESTATIONS OF SILICOSIS AMONG HARD GROUND WORKERS IN COAL MINES*

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OCCURRENCE

THE question of the occurrence of silicosis in coal miners is a very large one and one in which numerous and diverse factors are involved. That silicosis does occur, there can be no shadow of doubt. Its frequency among the whole mass of men employed underground would be very difficult to determine. Its incidence must vary according to the type of coal which is being mined, the geologic composition of the rock in the immediate vicinity of the seams of coal, the wetness or dryness of the conditions in any particular mine, and many other factors into which it is not proposed to enter.

The object of this paper is to draw attention to the following facts: (1) that there is one particular form of underground work which I think I can prove does directly, and (under the present conditions) inevitably, lead to an intense deposit in the lungs of dust which frequently contains a high percentage of silica; (2) that this leads to the condition known as silicosis of the lungs, very frequently accompanied by tuberculosis; and (3) that after a comparatively short period from the onset of symptoms the sufferer becomes permanently and totally disabled, and until his death drags out a miserable and breathless existence which is not even relieved by the knowledge that his dependents will receive any solatium in the form of compensation.

During the last thirteen years I have worked as tuberculosis officer in an area in which coal mining is the principal industry. It has been my practice to make careful inquiry into the working conditions of the patients. Gradually it has been borne in on me that I was with increasing frequency encountering a type of disease which was either not tuberculous or when proved to be tuberculous was of a type quite different in onset, course, and X-ray findings from what one may call the ordinary and straightforward case of pulmonary tuberculosis.

These cases were found to occur in men who had been working with machine drills in what are known as hard headings, and the type of case has often been so apparent that on glancing at a new patient for the first time, noting his breathlessness and general appearance, my first inquiry has been "How long have you been working on hard ground?"

It is intended that this paper shall mainly be a clinical study of a number

* Received for publication March 19, 1926.

of these cases, but it is necessary first to outline some of the salient features connected with this particular branch of the coal mining industry.

Drilling

In coal mining it frequently happens that the continuity of the seam of coal is broken in places by the occurrence of a "fault," and a tunnel has then to be driven through the rock adjoining the coal measures in order to reach the seam of coal at another level. Similar tunnels are often driven through the rock from one seam to another for ease of working. These tunnels are known as "hard headings," are usually 9 or 12 feet wide and about 6 feet high. For the last twenty to twenty-five years this work has been done by what are known as "machine" or "blast" drills. There are various types of drill, but in most of them the principle is the same. Compressed air is led from an engine some distance away through pipes to the hard heading. The drill is a massive apparatus which is supported at one end on the shoulder of the operator. The drill itself is 6 feet or so in length and is hollow. The compressed air pipe is connected to the machine drill which then rotates at a very high speed. Some of the air which drives the drill passes right through its hollow center to the bottom of the hole which is being bored and automatically blows the dust away from the hole. This dust is therefore blown forcibly backward into the face of the man operating the drill.

After the holes are drilled explosives are inserted and fired. Two or more drills may be in use simultaneously in the same heading which, being a *cul-de-sac*, is poorly ventilated. The noise made by each drill is something like the exhaust of a motorcycle; the dust in the air is so thick that the workers can hardly see one another, and added to this are the irritating fumes of explosives. The workers coming out of these headings are not at all like the familiar grimy colliers, but are white as millers, with hair, eyes, mouth, and nose choked with fine dust. Other men, working as laborers in these headings, removing the rock, etc., are almost as badly exposed to dust as the men operating the drills.

There are one or two other points connected with this work which are worthy of notice.

The work of driving hard headings is very rarely carried out by the personnel of the colliery itself, but is done by contractors who specialize in this form of work. The contractor engages to carry out the work at so much a yard. This leads directly to conditions which are altogether bad for the employee—the quicker that heading is pushed forward, the better for the contractor. The men are paid at a higher rate than for any other form of underground work; the contractor himself very often works with them; they hurry back soon after the shots have been fired and thus return into the irritation of the fumes of explosives as well as of the dust. If water were led to the rock face to be sprayed down the holes while boring was in progress, the dust would be diminished. Although conditions might then be better for the men, this precaution is very rarely taken, as the drills are thereby apt to become clogged and boring would be slower.

In the South African gold mines the use of hollow drills through which air *only* passes to the bottom of the hole being drilled is now prohibited by the Mining Regulations; in South Wales this is the type of drill almost invariably used. In South Africa water and air together are led through the center of the drill when the hollow type is used.

Another kind of drill which is sometimes used in these headings is a solid one which has a spiral ridge winding round and round its surface. This will bring back the dust more gently and with less dissemination in the air, but men who have worked with these drills say that they are very apt to become clogged and are not widely used.

The men rarely take any measures to protect themselves from the dust; occasionally they wear a handkerchief or wet sponge over the mouth or nose, but respirators are not worn with any regularity. At the best, it is well known that respirators are not efficient. The particles of silica which cause most trouble are those of less than 4 microns in diameter, particularly those of 1 or 2 microns. Tests carried out by the South African committee on respirators showed that about 50 per cent. of the dust particles could be trapped in a respirator, but all the particles which got through were about 1 micron in diameter and, therefore, dangerous.

In the intervals between working on hard headings, many of these men work on the coal or as "sinkers" of new pits. Machine drills are also used in "sinking" but this work appears to be much less dangerous—the conditions are more open to the air, there is almost always water in the shaft, and the holes are bored directly downward—and although I have never been down to see this work in progress the men tell me that it is not as a rule dusty.

Relation to Geologic Formation

My own observations have led me to the conclusion that the incidence of cases of silicosis varies with the geologic formations encountered. In the northern part of this district the collieries are practically all working the same seams of coal and will encounter similar rocks. At the western end, however, is the large anthracite field. The present paper deals with the clinical details of twenty-two cases which have come under my observation comparatively recently. No less than twenty of them come from a small area of which Cymmer may be considered the center, and which includes the head of the Neath Valley—the Afan Valley—Maesteg and Ogmore Vale. So far, in spite of the great developments in the anthracite coal field in the last fifteen years and the driving of a large number of hard headings there, my cases do not include a single one in a worker on the anthracite field.

At a later date it is my hope to get detailed analyses of the dusts from various areas. Analyses are already available of samples of dust taken from four headings in the Cymmer and Glyn Neath area which show 61.6 per cent. of silica in the Cymmer headings, and 57.7 and 56.7 per cent. in two others at Glyn Neath.

A mining engineer tells me that in the anthracite field the rock encountered will be of a softer and more shaly

character. This may explain the marked variation of incidence in different localities; but until further analyses are available this is only put forward as a suggestion. It would appear clear, however, that men working in headings under the terrible conditions which have been described and exposed to dust containing some 60 per cent. of silica can hardly expect to escape an intense deposit of silica dust in the lungs.

Dusting

One other observation must be made before turning to the clinical side of the picture, and that refers to the practice of what is known as "dusting" in coal mines. It has been found that if certain kinds of stone dust are scattered or "dusted" all through the coal workings there is a diminished liability to explosions—and the underground workings nowadays look as if they had been whitewashed because this dust has been scattered all over the floor, walls, and roof of the workings. The dust originally used was a slate dust, rich in alumina and with a very low silica content. It appears, however, that many collieries now make their own dust—that the rock of any kind which is brought up from below is crushed at the pithead and sent down again for dusting the workings. Here seems to be another source of danger to the miner, especially to the men employed in scattering the dust, if rock such as comes from our local hard headings containing 60 per cent. of silica is used for this purpose.

It is probably too early to judge whether the dusting of mines is leading to ill health in the workers, but the interesting remark was made by the veterinary surgeon at one of the local collieries that since dusting had been introduced there was a considerable increase in the number of broken-winded horses.

Clinical Manifestations

From the conditions of work we now pass to a consideration of the symptoms, signs, and other clinical features observed in a series of twenty-two patients, all of whom have worked for various periods with machine drills on hard ground.

Duration of Exposure and Latent Period

It is very important to remember that the onset of symptoms is usually slow and insidious. The slowly progressing fibrous changes in the lungs do not as a rule produce symptoms for a considerable number of years; thus there is almost always a latent period of several years between the commencement of exposure and the onset of symptoms.

In this series of cases the ages of the men when first seen varied from 32 to 60, the duration of exposure to stone dust varied from 12 months to 25 years, and the latent period which elapsed from the commencement of work to the onset of symptoms varied from $3\frac{1}{2}$ to 22 years. The averages were as follows: age when first seen, 43.7 years; duration of exposure, 10.8 years; period before symptoms developed, 10.6 years.

It has been found in South Africa that the average time elapsing between the commencement of exposure and the onset of symptoms is nine and one-half years. The figure in my own cases is ten and one-half years.

These figures are quite near enough to draw a parallel, especially as in South Africa the men are closely watched and are encouraged to report early symptoms, whereas the bulk of the workers in my series have been unaware of their danger and when first seen were advanced cases and probably had suffered from slight symptoms for a longer period than they were prepared to admit. Their exposure, too, in many cases had been intermittent, with a few months of work on hard ground and then back to coal again. In no case where the exposure had been regular and severe were symptoms delayed longer than thirteen years. Some of the men, moreover, had changed their occupation for various reasons quite apart from health, but in due course the inevitable dyspnea came on. One man, for instance, worked eight years regularly with machine drills, from 1906 to 1914. He then joined the army, was passed as A 1, but in spite of this open air life dyspnea came on in 1918, and from then until his death six years later his illness was a typical case of silicosis.

Initial Symptoms

Dyspnea.—The first and often the most marked symptom throughout the whole course of the disease is dyspnea.

In fifteen out of these twenty-two cases dyspnea was given as the first sign of ill health, and although the others gave pleurisy, winter cough, or pneumonia as the mode of onset, all mentioned dyspnea as following on immediately from the initial complaint.

In most cases it is probable that slight dyspnea exists for some time before the patient really complains of it, and it is when he develops an intercurrent catarrh that it becomes really noticeable and leads to his seeking advice.

It is rather remarkable that cyanosis is rarely present, even when the dyspnea is very pronounced. It was observed in only two patients—one of whom had a small accidental pneumothorax.

Pleurisy.—Pleurisy was given as the initial symptom in four cases, three of which proved eventually to be cases of silicosis plus tuberculosis. Apart from pleurisy as an indication of a probable tuberculous infection, many of the patients suffer from transient pleuritic attacks, often with a coarse leathery rub. In no case has there been pleurisy with effusion.

Cough.—Cough was given as the initial symptom in only two cases—a slight winter cough with scanty expectoration getting worse for two or three years, with gradually increasing dyspnea.

Later Symptoms

Dyspnea.—In the further progress of the disease, dyspnea continues to dominate all the other symptoms.

Cough.—Cough with more or less expectoration is recorded in every one of these cases, but does not as a rule trouble the patient to anything like the extent that it does the average patient with straightforward tuberculosis. It is typically short and paroxysmal, occurring mostly at night and in the morning.

Sputum.—The sputum is usually slight. This has been a striking feature in the series of cases. In

only four out of twenty-two was it copious, and these were all cases which were positive for tubercle bacilli. In the rest the expectoration usually consisted of small, hard, whitish lumps of a firm gelatinous consistency. It often happened, particularly in the earlier cases of the series, that on examination a man was found to have marked signs which were unhesitatingly considered to be due to tuberculosis—only to find that repeated sputum examinations were negative and invariably recorded by the laboratory as "very poor specimens." Probably many of these cases with negative sputum are tuberculous, but this point will be dealt with later on in this paper. In the cases where the sputum is larger in amount it may be very frothy or contain quantities of black dust, but it is rarely of a typical nummular tuberculous character, even when tubercle bacilli are found.

Loss of Weight.—Loss of weight occurred in every case, usually to a considerable extent.

Lassitude and General Weakness.—This symptom was present in most cases but is not a pronounced feature in the early stages.

Hemoptysis.—Hemoptysis occurred in only four cases and was slight in amount except in one of the tuberculous patients who died from a fatal hemorrhage.

Spontaneous Pneumothorax.—This was observed in only one case. It was slight and limited to the apex, and was not suspected until revealed by X-ray examination.

Physical Signs

The present series of cases unfortunately is composed almost entirely of men whose condition was relatively far advanced when first seen. They have all been referred for examination as cases of suspected tuberculosis, and by the time the average busy colliery doctor has his suspicions seriously aroused the patient is rarely in an early state of disease.

It follows, then, that the physical signs observed in these cases are for the most part extensive and obvious. In discussing this question Dr. Middleton (1) says: "Silicosis is a disease only according to the degree in which it departs from the accepted normal of adult life . . . . Only by collecting information regarding the slightly affected cases can knowledge be arrived at as to what degree of divergence from normal is to be regarded as requiring precautionary measures, such as removal from an industry, and what may safely be allowed to pass." It is my hope in future to examine many more of these workers before symptoms develop, and thus to collect information about the earlier manifestations of silicosis.

In general, the signs observed in this series of cases may be said to vary from those of slight emphysema to those presenting a condition closely resembling advanced chronic pulmonary tuberculosis.

Facies and General Appearance.—More or less breathlessness is often apparent even on talking to the patient and there is frequently a rather typical weather beaten type of face, deeply lined, especially from the nose to the angle of the mouth, but the patient is not so pale or ill looking as the average case of tuberculosis.

The chest wall is more or less fixed, with supraclavicular hollowing, and there is overaction of the extraordinary muscles of inspiration. General nutrition is poor and the muscles flabby.

Areas of dulness were observed in every case, the most striking feature being a large fan shaped area of dulness spreading out from one or both roots. This may be the only area of dulness found but is often associated with apical dulness as well. Thus, in these twenty-two cases the dulness observed was:

Area	*No. of Cases*	
One apex	2	
Both apices	4	
Roots only	3	17
Roots and apices	14	
Bases	4	

The basal dulness was chiefly in pleuritic cases, and apical or root dulness was also present in these cases. The striking feature is that in seventeen out of twenty-two cases these large oval areas of interscapular dulness could be easily demonstrated.

The breath sounds are usually weak toward the bases, and in some cases all over the chest. In the root areas in almost every case the breath sounds were bronchial or bronchovesicular. Thus in nineteen cases the breath sounds at the base were weak, and in seventeen, breath sounds of a more or less bronchial type were noted in the interscapular areas.

Adventitious sounds are sometimes entirely absent; at other times the chest is full of high-pitched rhonchi, especially if there is any intercurrent catarrh.

Crepitations may be heard at the apex or base but the most typical moist sounds are heard in the root areas in those cases where root dulness is present, and consist of fine crepitations and numerous very fine sibilant rhonchi which seem to convey very definitely a sense of obstruction and moisture in the finer bronchioles.

Temperature Records.—A remarkable feature of these cases, even those in which there is definite evidence of superadded tuberculosis, is the almost complete absence of fever. Time and again a case has presented extensive dulness, areas of bronchial breathing and numerous moist sounds, which would have been accompanied by persistent pyrexia if the case had been one of straightforward tuberculosis; but on observation in hospital for several months the case has been continuously afebrile. Nine patients in this series have been in hospital for periods from two to six months.

Of four cases in which no tubercle bacilli were ever found in spite of repeated examinations, three were absolutely afebrile, the other only twice had a temperature of 99° in three months' observation. The remaining five cases all had tubercle bacilli in the sputum and extensive signs; three of these were continuously afebrile and neither of the others ever had a temperature over 99°. It would appear obvious that a temperature record is not sufficient to enable one to draw the difficult distinction between simple silicosis and silicosis plus tuberculosis.

Progress and Course.—The progress of every one of the twenty-two cases under review has been one of steady decline. Dyspnea persists and increases, cough becomes worse, and

there is progressive weakness and loss of weight. In the earlier years after the onset of symptoms there onset, no improvement in dyspnea and patient more subject to bronchitic attacks but doing light work.

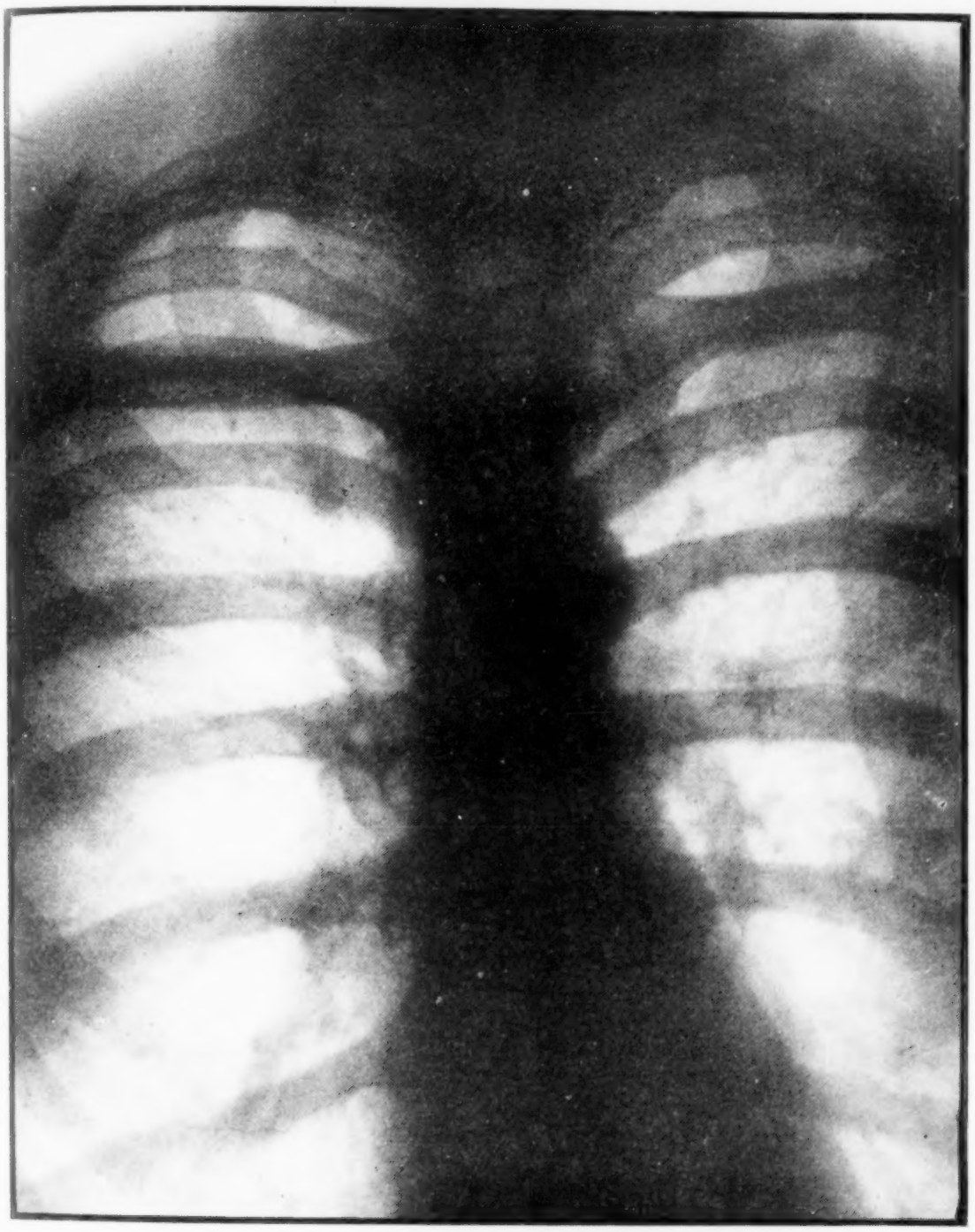

FIG. 1.—Silicosis stage 1, showing reticulation and slight nodulation; heart good shape. A. P., worked ten years "off and on" with machine drills; dyspnea started after eight years' exposure; sputum negative.

may be a period when the condition appears to be unchanged or to get worse only very slowly. Thus in some of the cases which appear to be pure silicosis the report is as follows: two years after onset, patient fairly well and regularly employed on light work; four years after onset, patient slowly losing weight and too dyspneic to work; and eighteen months after

In some cases, particularly those in which the exposure has been very severe, the fatal termination has been rapid, after fourteen months in one and fifteen months in another. Apart from the intervention of tuberculosis, the termination is like that of chronic bronchitis, with respiratory and cardiac failure.

Eleven patients in this series have

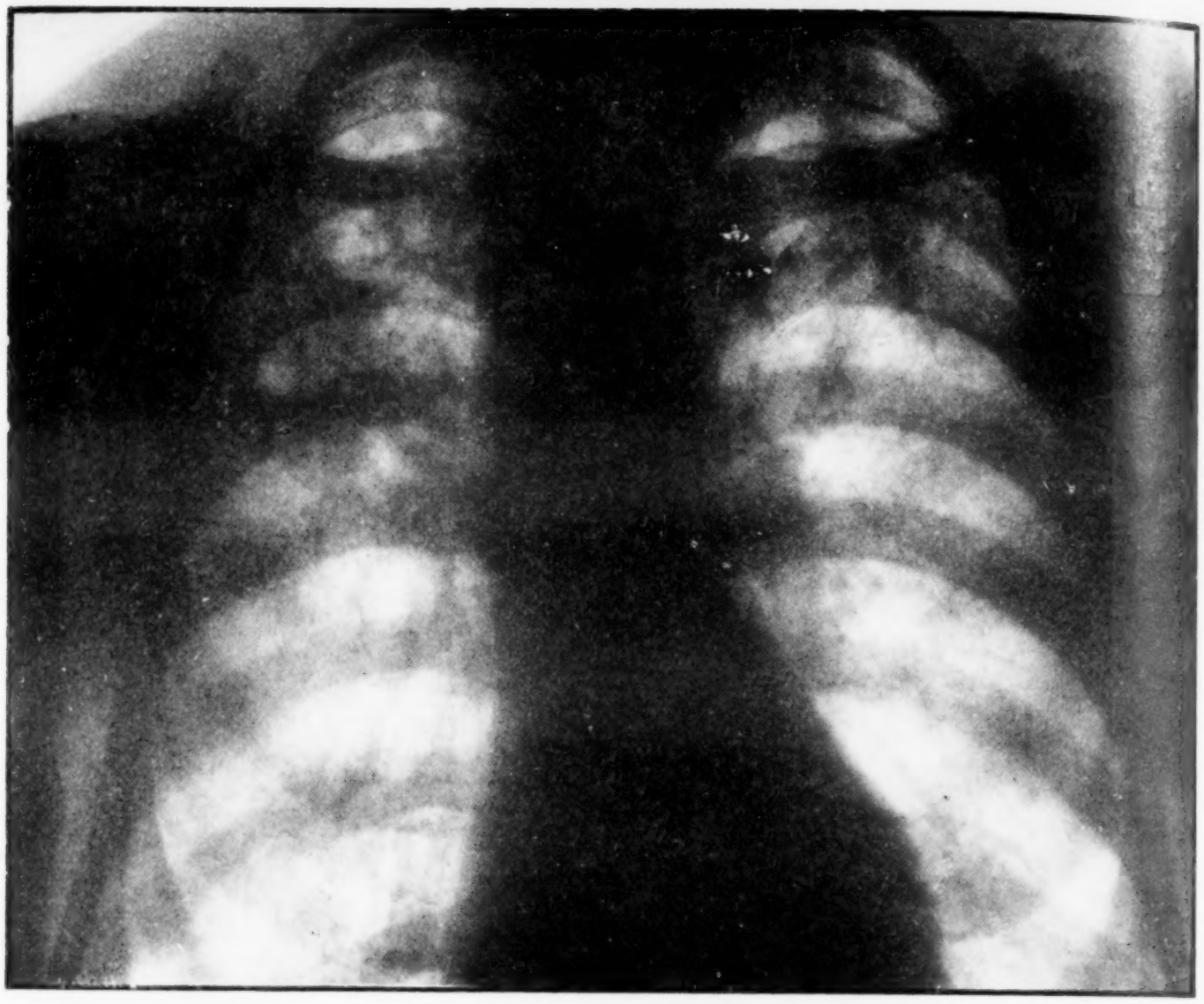

FIG. 2.—Silicosis stage 2, showing fine nodulation with probably slight tuberculous deposit at apices; heart good shape. W. D., worked ten years with machine drills; pleurisy and dyspnea after nine years' exposure; sputum negative.

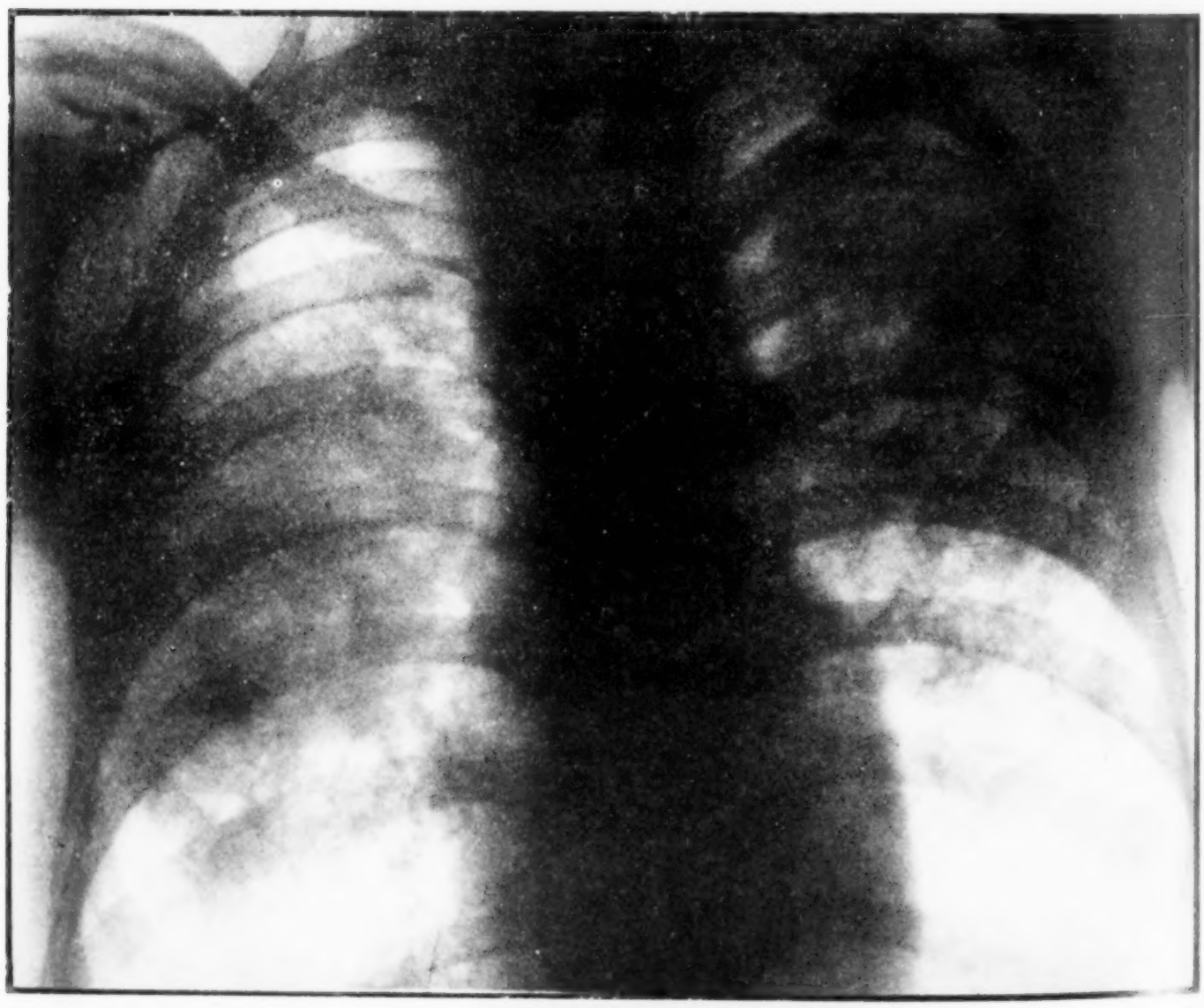

FIG. 3.—Silicosis stage 2, showing fine nodulation, plus tuberculosis indicated by areas of clouding, by dense aggregation, and by vertical heart. R. E. H., worked several years regularly with machine drills; dyspnea after six years' exposure, with clinical onset of tuberculosis four years later; sputum once positive in several examinations.

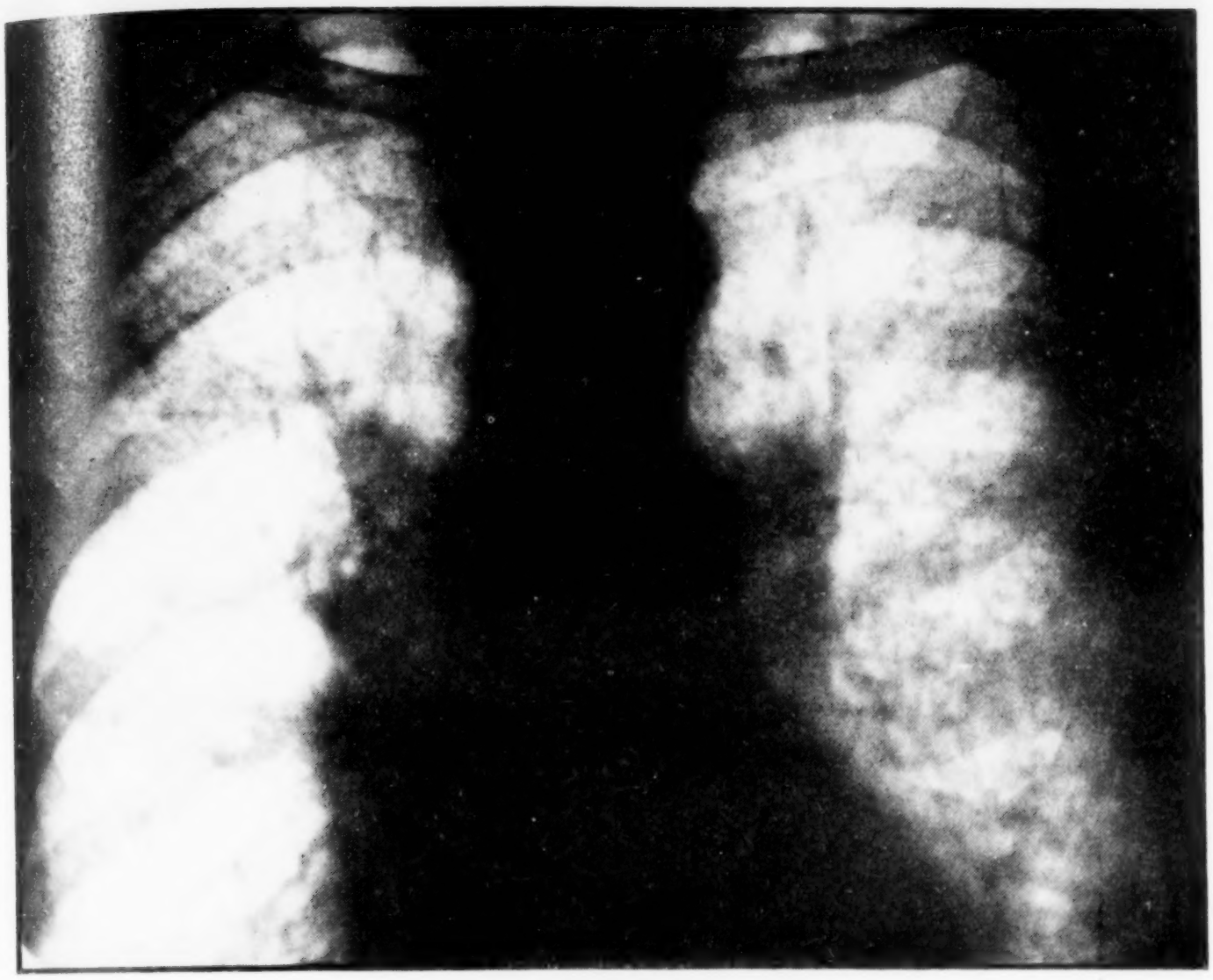

FIG. 4.—Silicosis stage 3, showing coarse nodulation; heart good shape. J. W., worked fifteen years "off and on" with machine drills; dyspnea started one year after giving up this work, or sixteen years from first exposure; sputum negative.

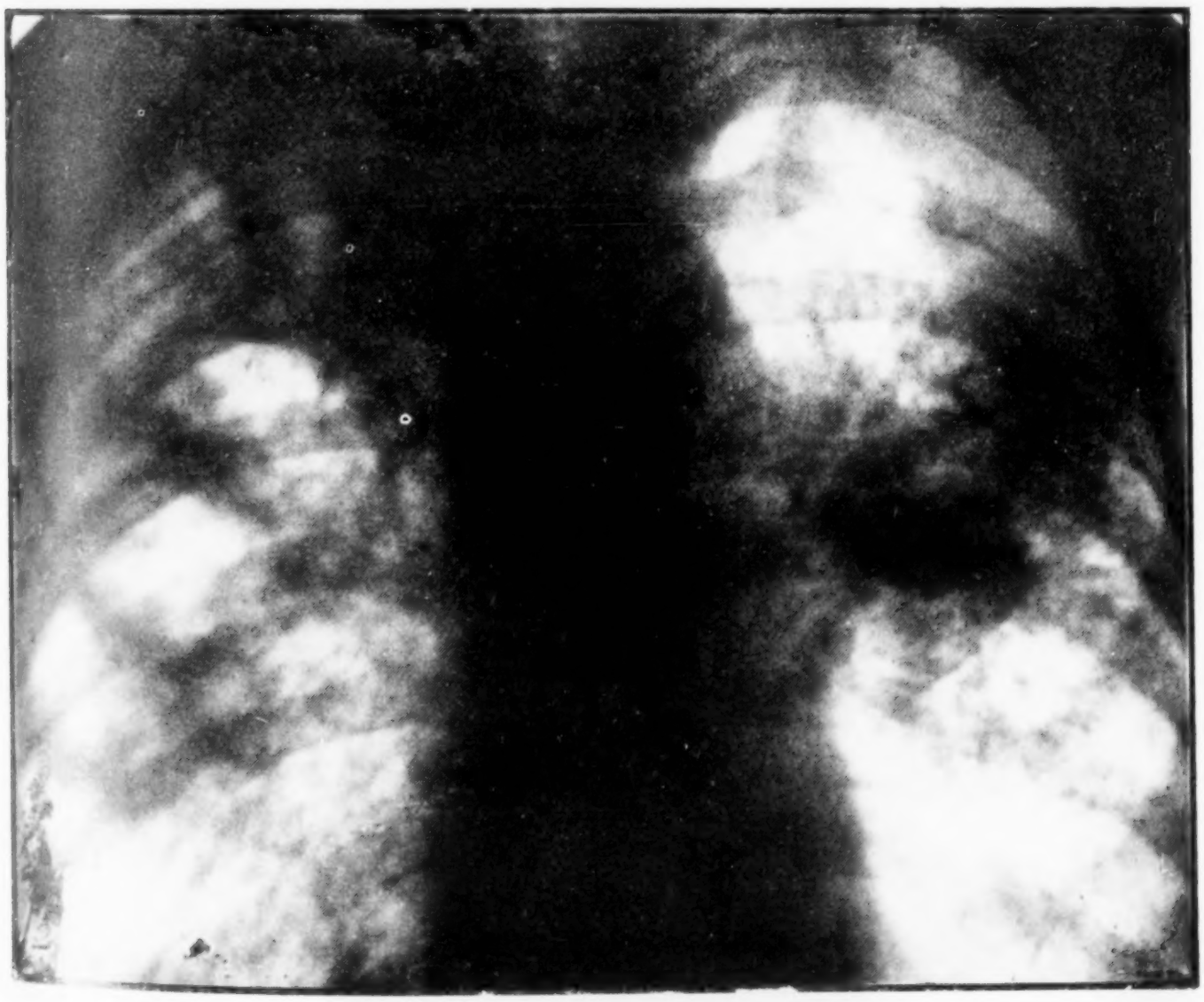

FIG. [illegible] Silicosis stage 3, showing coarse nodulation, plus tuberculosis indicated by aggrega[illegible] masses in both lungs and by vertical heart. E. W. J., worked six years con[illegible] with machine drills; dyspnea after five and a half years; sputum positive.

already died. The average duration of life after the onset of symptoms was four years in the cases which did not show positive evidence of tuberculosis, and just under four years in those in which tubercle bacilli were found.

It is a matter of great regret to me that so far it has been possible to get a postmortem examination in only one case.

Radiography

The value of radiography in the diagnosis of silicosis and silicosis plus tuberculosis has been left to a late stage of this paper because it appears from the very positive statements made by the experts on the staff of the South African Miners' Phthisis Bureau that by the use of X-rays alone is it possible to determine with some degree of accuracy the extent of silicosis and

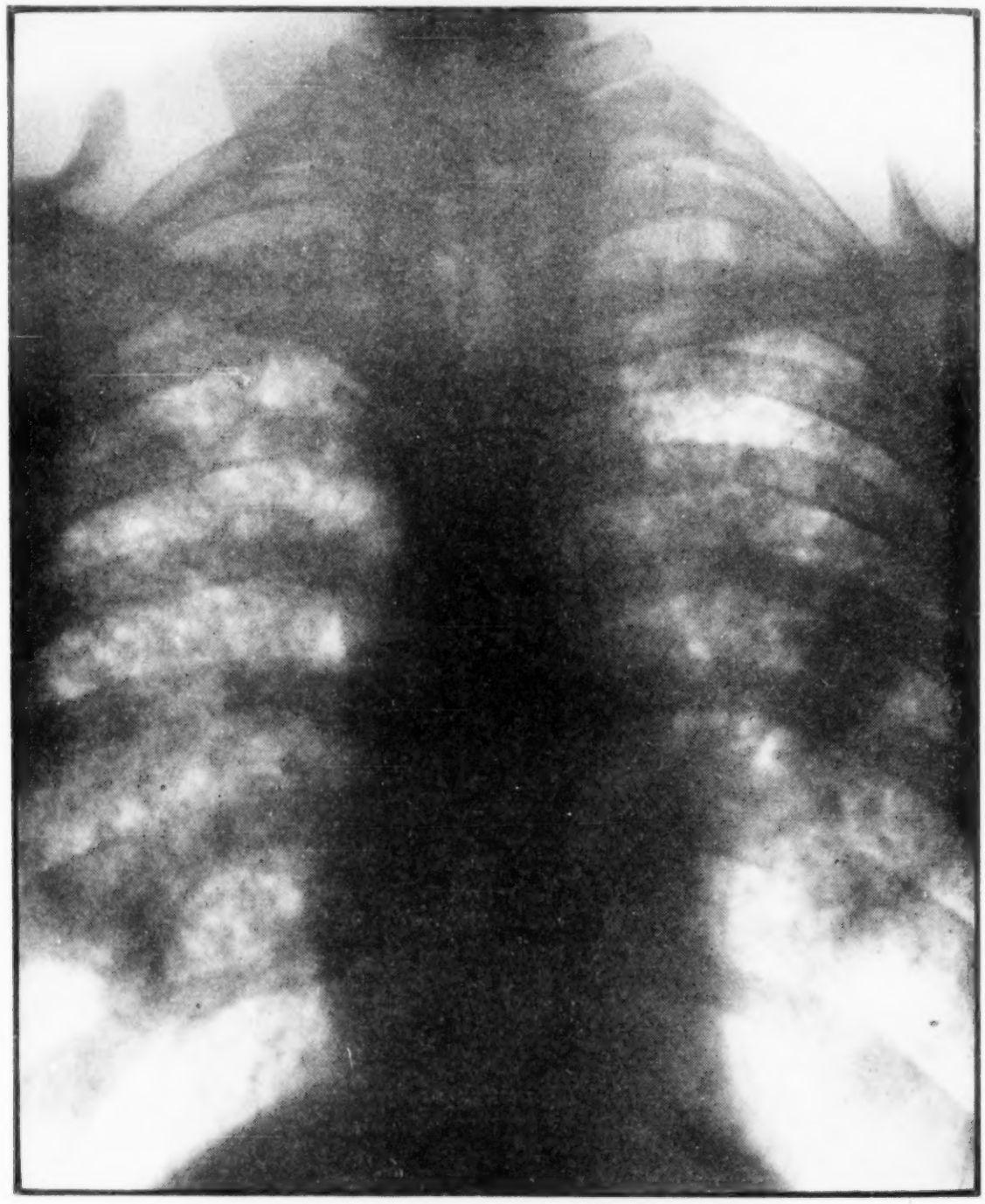

FIG. 6.—Silicosis stage 3, showing coarse nodulation, plus tuberculosis indicated by clouding, aggregation, and vertical heart. H. E., worked one year only with machine drills, ten years ago; dyspnea eight years later; sputum negative thirteen times, including guinea-pig inoculation.

the onset of tuberculosis in silicotic subjects.

Dr. Watkins-Pitchford, in his annual report of the work of the Bureau for 1923 (2), states: "It can now be alleged that if a technically satisfactory radiograph, taken within a few months of death, has not disclosed the earliest signs of simple silicosis these will not be discovered by the pathologist. It has indeed been remarked, on more than one occasion of late, that radiographic evidence appeared to be more pronounced than that afforded by subsequent post-mortem examination."

In South Africa the opportunities for arriving at dogmatic conclusions such as this are unequaled. Every year something like 25,000 radiographs are taken, and there is a continuous X-ray record of each worker. All findings are further checked by a postmortem examination in every fatal case. The report further deals with the value of X-rays in detecting closed tuberculosis and of the "vertical heart" on which they lay great stress as being suggestive of a tuberculous infection.

Dr. Steuart (3) who carries out the X-ray work has described the typical appearances in an article based on the examination of 200,000 radiographs. With regard to the "vertical heart" he states: "Whatever be the explanation of the phenomenon, it is certain that such individuals, when working underground, are more prone to develop silicosis, and/or pulmonary tuberculosis, than are other miners."

Steuart describes three X-ray stages in the development of silicosis, with a fourth stage or series of changes which indicate added tuberculosis. The stages are briefly as follows:

1. *Reticulation.* A general increase in fibrous tissue, bringing out gradually the shadows of the smaller tubes so that a linear network appears throughout both lungs, more marked as a rule toward the right base.

2. *Nodulation.* There is an apparent segmentation of the linear network into closely aggregated miliary nodules.

3. *Coarse Nodulation.* The nodules increase in size but diminish in number—remaining *clearly cut.*

4. If tuberculosis supervenes, the nodules become still larger and *ill-defined* or floccular, while areas of clouding or coarse aggregation appear in the hitherto uniformly mottled lung shadow.

In preparing this paper, my twenty-two cases were classified on clinical grounds into three groups:

1. Twelve cases of silicosis which (apart from X-ray examination) failed to show any evidence of tuberculosis.

2. Six cases of silicosis with tuberculosis in which tubercle bacilli had at one time or another been demonstrated.

3. Four "doubtful" cases, in which no tubercle bacilli had ever been found, even after as many as fourteen examinations and a negative guinea-pig test in one case, but all of which were thought probably to be tuberculous as well as silicotic.

When these clinical groupings are reviewed in the light of the South African experience, the picture is changed:

1. The twelve supposed pure sili-

cotics become six cases of silicosis and six of silicosis plus tuberculosis.

2. The next six cases which were known to have tubercle bacilli were all confirmed as such by the South African standards.

3. The four "doubtful" cases all become cases of silicosis plus tuberculosis.

Thus out of the series of twenty-two, sixteen must on X-ray findings be considered to have superadded tuberculosis.

Silicosis Plus Tuberculosis

These remarks lead directly to some observations on the question of the double disease—silicosis plus tuberculosis. Is the tuberculosis primary and merely changed in type by the presence of silica, or does silicosis generally exist first and by its presence lead to an increased susceptibility to tuberculosis which takes on a particularly slow and nontoxic type of evolution?

The South African observers are rather inclined to suggest a primary tuberculosis. Thus Dr. Watkins-Pitchford (2) in discussing simple silicosis suggests that the development of the silicotic nodules to more than microscopic dimensions "appears to be the result, in at least many instances, of a peculiar form of tuberculous infection generalised throughout the lung; although, unless other and more orthodox indications of tuberculosis subsequently appear, the condition is still considered to be one of simple silicosis." Later in the same report he says that it appears probable that "the great majority of cases of simple silicosis owe their earliest detectable development to the presence of an occult tuberculous element . . . . this element would go far to explain the relatively slight effect which change of environment appears to have upon the subsequent history of the majority of cases."

It is not proposed at this stage to go with any detail into the bacteriologic researches which have been carried out on this subject. Gye and Purdy (4) and Gye and Kettle (5) have clearly demonstrated the antibactericidal action of silica in relation to the tubercle bacillus, and some experiments carried out by Professor Cummins (6) appear to indicate that this action is due to interference with "complement." Probably the truth lies between the two, and it is my impression from the purely clinical standpoint that, whereas tuberculosis may in some cases be primary, it is much more frequently a superadded infection.

One tragedy which has come to my notice illustrates the danger to these workers and is most likely an example of superadded tuberculosis.

Some three and one-half years ago accompanied by Professor Cummins I visited a hard heading at Glyncorrwg. Two shifts of men were being used to drive a heading through some 300 yards of hard rock—three men boring on each shift. The dust was intense, so thick that one could see only about a yard when drilling was in progress. One of the men recognized me and I found out that he was a man I had recently examined and who was probably tuberculous but at that time one sputum examination had been negative. About six months later I saw this man again when he had marked

signs and numerous tubercle bacilli in the sputum. He had been working all along in the same heading coughing and spitting out tubercle bacilli. He died about a year later, the first of these six men to succumb. The sixth and last of them died two and one-half years later from silicosis and tuberculosis.

Three and a half years ago, all these men were, as far as they knew, in good health, though no doubt some were already silicotic. They were all in the prime of life. Further comment would appear superfluous.

Prevention

Incidents such as the one just quoted could be multiplied, but what has been said is surely sufficient to support the plea that the conditions of this branch of the mining industry are such as to call for immediate inquiry and government intervention. In South Africa the results obtained have been marvellous. It is true that the number of men in this country employed for more or less whole time on hard ground is small, but that fact is not sufficient to justify apathy.

Some inquiries as to the number of men employed on hard ground were made from a local Member of Parliament who is closely connected with the Miners Federation. He replied: "It is very difficult to find out the exact number of men engaged on this work. At Duffryn Rhondda Pits where 1,200 men were employed only 18 worked on hard ground. At Glyncorrwg and Abergwynfi about 30 men worked on this ground out of 1,000. Where less than 100 are employed at a colliery generally you have no hard ground men, but in large collieries they vary from 8 to 30.

"In Cwmavon and Tonmawr where there are many faults in the strata a few men are employed for 4 to 5 weeks, 5 or 6 times a year, in which case they would work about 6 months on coal and 6 months on hard ground."

Recommendations

In conclusion it is not presumed to lay down what scheme of prevention should be adopted, but the following would appear to be reasonable suggestions:

1. That all men employed on hard ground should be registered.
2. That prior to registration the men should undergo a rigorous medical examination including an X-ray examination.
3. The men should further be subject to annual reexamination.
4. There should be rigorous exclusion from the work of anyone suffering from or suspected of tuberculosis.
5. Dry drilling should be abolished by statute.
6. Regulations of a protective nature should be enforced, such as the use of the best possible type of respirator and the leading of water to the holes during drilling.

Further investigation may lead to some suggestions as to the type of dust to be used for antiexplosive purposes.

Until adequate measures on these or similar lines are taken, nothing can prevent the occurrence with distressing frequency of cases such as the present communication has attempted to describe.

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EXPOSURE TO SILICA DUST WITHOUT THE OCCURRENCE OF SILICOSIS*

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INTRODUCTION

THE craft of the sculptor and the stone cutter has existed from the earliest glimmer of dawning human history; from the time when paleolithic man scratched images of his animal contemporaries on the walls of his cave dwellings, working with hard chipped flints upon the softer stone that forms the pages of our earliest manuscripts. We owe to the stone cutter, and to the potter and tile maker, nearly all of the little knowledge we possess of the civilization of our early forefathers. It would seem, indeed, as if the members of the craft, throughout the ages, felt that a great responsibility rested upon them; that they were concerned with work which would endure, and that hence they took great precautions to insure that the members of their guild should be worthy of the trust which was reposed in them. These traditions persist to this day. The craft is still, to a great extent, hereditary; the sons are apprenticed to the fathers, and the stonemasons form a clearly defined group or guild of handicraftsmen, possessing a unique historical tradition of practical and disciplined democracy. We are told, for example, that at the building of the Parthenon, the architects, stone cutters, masons, and draftsmen were all free men; and all received the same remuneration for their services. A Derbyshire stonemason of today, visiting the ruins of Le Baux or Petra—the "rose red city, half as old as time"—or Luxor, or the Acropolis, will find himself amidst the work of "his ain folk;" will note the cuts of the chisel; look out for the individual "mason's mark;" appreciate each difficulty his fellow craftsman had to deal with; and feel as if, from the past ages,

> Someone there stole forth a hand
> To draw a brother to his side.[1]

We need not wonder why it was that from this great guild grew the famous secret oath bound brotherhood of democratic mutual assistance, whose symbol is the interlocked compasses and square, and whose lodges are found throughout the world.

It does not appear that the stonemasons' disease was recognized in classical or early medieval times. This may be, to some extent, explained by the fact that silica rocks were little worked by the ancients.

The Egyptian stone cutter worked in many kinds of stone. Sandstone was quarried at Silsileh, the finer

*Received for publication June 17, 1926.

[1] Flecker, J. E.: Collected Poems.

and harder stones such as alabaster, porphyry, diorite, etc., at Hammabat and Habnub, granite at the first cataract, and limestone all along the Nile valley, which cuts through the North African plateau of nummulitic limestone from the first cataract to the delta.

Khasikhemui's tomb—"the earliest stonemasonry structure known in the history of man" (Professor Breasted)—built not later than B.C. 3,000, is of hewn limestone. The great pyramid (Cheops), built by King Khufu of the fourth dynasty about B.C. 2,900, and the greater part of the lesser pyramids, are of limestone: the surrounding pavements are of limestone or basalt.

The sepulchral chambers within the pyramids have plug blocks and portcullises of granite, and part of the casing of the pyramid of Khafra, whose reign commenced in B.C. 2,869, is of the same material.

The earliest Egyptian altars were hewn from one block of granite, and the "palm tree" columns of the fifth dynasty temples—"the earliest known columns in the history of architecture" (B.C. 2,750 to 2,625)—are granite monoliths, carved to represent palm trees or bundles of papyrus stalks. The "pylon" towers of the later temples are of limestone or sandstone.

The Egyptian obelisks are granite monoliths from the first cataract. The London obelisk, Cleopatra's Needle, is one of a pair erected by Thutmosis III. at Heliopolis in B.C. 1,461. Its companion is in New York. The tallest of the obelisks, that erected by Thutmosis IV. at Karnak, in B.C. 1,400, is 105½ feet high, and now stands before the Lateran in Rome.

The colossi are chiefly of limestone or granite, but at least two, the famous "Memnon" colossi of Amenhotep III. (B.C. 1,411 to 1,375) are of gritstone, from a quarry near the present site of Cairo. Standing over 60 feet in height on the western plain of Thebes, they are much weathered, and the sound made by the wind playing through their crevices and cavities has led to their being known as the "singing colossi."

Similarly the buildings of classical Greece and Rome were chiefly either in marble or in brick. Such references to "pneumonokoniosis" (a term coined by Zenker) as are made by Hippocrates and Pliny refer to mining and metal digging rather than to stone cutting. Georgius Agricola (cited in (1)), in 1557, wrote of the use of respirators in mines, to prevent dust inhalation, and tells us that the miners in the Carpathian mountains suffered so severely that women could be found who had married as many as seven husbands! Ramazzini, in 1703, published a treatise, "The Diseases of Tradesmen," in which he referred to the prevalence of phthisis in stone cutters, masons, ore miners, potters, bakers, etc. This appears to be the first mention of stone cutters and masons in this connection.

Thackrah (2), of Leeds, in 1832, pointed out that men are injured when working (lead) ore in sandstone, but are not inconvenienced when the ore is in limestone—a fundamental observation which has since been amply confirmed.

True stonemasons' disease, in Derbyshire, was described by Barwise in 1913 (3), when he pointed out how the male phthisis mortality of the

County was associated with the geology of the County, which in its turn determined the occupations of the males, either as agriculturists, or coal miners, or workers on millstone grit.

My experience as Tuberculosis officer to the Derbyshire County Council enables me to say that the disease nowise differs from silicosis seen elsewhere; it is a pulmonary fibrosis and sclerosis, due to the deposit of fine silica dust throughout the parenchyma of the lungs; this dust reaches the lungs by inhalation of the fine particles struck off the stone in the various processes of dressing, sawing, etc. The changes increase with the hardness of the stone and the purity of its silica content.

The best Derbyshire millstone grit contains over 98 per cent. of pure silica, and is one of the toughest and most durable of stones known to man; being, in contradistinction to the older carboniferous rock, particularly resistant to the action of acids. It is extensively used in the manufacture of pulping stones for wood pulping mills. Most of the Tudor halls, churches, and manor houses of Derbyshire are built of millstone grit, as are the towns of Buxton and Matlock.

Silicosis among stone workers has been so accurately described by others, notably for Derbyshire stone workers by Nicholson (4), that further description is here unnecessary. There has, however, come under my notice an interesting group of men who are exposed in their work to silica dust, but who manifest no symptoms of silicosis, nor any undue mortality from phthisis. These men make ganister bricks, "a term which appears to be applied to bricks composed of crushed ganister rock, or silica sand, as found in Derbyshire and Staffordshire, mixed with varying amounts of fireclay or other local clays" (5). Such bricks are "refractory," that is to say, they withstand fairly high temperatures without melting; but they are not so highly refractory as silica bricks made from crushed quartzite, containing no clay, bonded together with lime.

The importance of the absence of silicosis among these men may be appreciated by reviewing briefly the action of inorganic dusts in the lungs.

Action of Inorganic Dusts

There are enormous differences in the effects produced in the lungs by the inhalation of different forms of inorganic dust. Some are, of course, protoplasmic poisons, *e.g.*, lead, arsenic, antimony. Others, like the dust from soft coal, infiltrate the lung tissue without causing much inconvenience or many symptoms of disease. The dust of chalk, oolite, gypsum, marble, limestone, and the whole calcareous group, is comparatively harmless. When signs of pulmonary dust fibrosis occur in limestone workers it will invariably be found that there are veins of silica rock, *e.g.*, chert, flint, etc., embedded through the limestone. Miners in anthracitic and hard coal, working on the coal face, develop in time a moderate miners' asthma; and here again it appears to be the free silica contained in the rock overlying or surrounding the hard coal seams, rather than the coal dust, which is the causative agent.

Argillaceous rocks, aluminates, such

as those of Portland cement; hematite and other iron ores; clay, loam, and soil dusts are in themselves harmless. They may, of course, contain dangerous micro-organisms. The dust from carborundum and emery wheels used in the cutlery trade is far less injurious than that thrown off from sandstone wheels.

Certain inorganic dusts, when inhaled, have peculiar effects which may be briefly referred to. The dust produced in grinding basic slag, for the production of fertilizers, is, according to Oliver, capable of setting up very severe pulmonary disease of the type of acute croupous pneumonia. The dust of red oxide of iron, used by glass polishers, and of hematite, when inhaled, may produce a permanent brick red pigmentation of the lungs without any corresponding impairment of health (1).

Of all forms of inorganic dust, the only one which is responsible for definite pulmonary fibrosis is the dust formed by free silica in minute subdivision; or, in other words, the dust of the material in which the Derbyshire stonemason works.

Free silica occurs, of course, in other forms beside millstone grit. Quartz, flint, chert, and ganister are all practically pure silica, granite is an aggregate of quartz, feldspar, and mica, and the dust from all these formations is capable of producing silicosis in those exposed to it. From quartz dust we get the deadly silicosis of the South African miners, from granite dust the milder silicosis of the Cornish miners and the Aberdeen masons. From flint and chert dusts come flint knappers' silicosis and potters' asthma. Ganister dust causes silicosis in the miners who dig it out of the earth, and in the makers of silica bricks, crucibles, abrasive soaps, and others who use powdered ganister in industry. From the sandstones and millstone grit come grinders' rot and stonemasons' disease.

All these diseases are essentially one, the characteristics of which are (1) a progressive pulmonary fibrosis, with accompanying increasing dyspnea, and (2) a tendency to develop and to succumb rapidly to pulmonary tuberculosis, in late middle life.

Silica Brickmakers

In the high limestone plateau of Derbyshire there occur pockets of very fine silica sand, still somewhat a geologic puzzle. Under high powers of the microscope, the particles of this sand appear as irregular pebbles, without sharp spicules, surrounded by an earthy coating. Analyses of the material are given below.

For many years this sand has been used in the manufacture of silica bricks. After molding, the bricks are fired in a kiln, and the dust arising from trimming and polishing the finished articles is considerable. Indeed the process of making these bricks is a dusty one. The clothes of the workmen, when beaten, emit clouds of dust. The hands of the men and women employed in the various processes have the white appearance and silky feel characteristically found in silica workers. Over 200 workers are regularly employed in the industry; but up to 1922, no case of silicosis had been reported among the employees, and the tuberculosis death rate among them was well below the general death rate of Great Britain.

About 6 miles away from these brickworks are the Stanton and Darley Dale gritstone quarries. The stone dressers in these works notoriously suffered from silicosis (3), and it was not unusual to find that workmen left the stone dressing industry to take up work in the brickworks, as the latter was recognized as a healthier occupation.

In the course of the writer's examinations of these brickmakers, under the Refractories Industries regulations, a certain number of cases of silicosis were found. Inquiry into the previous history of these affected individuals elicited the startling fact that practically every one had formerly worked as a gritstone dresser, and that the condition was attributable rather to the stone dust than to the dust from the silica sand.

Thus out of 176 males employed at one brickworks in 1922, 18 had formerly worked on millstone grit (14 for over five years and 1 for as long as thirty years). The pulmonary condition of 8 of these men, or 44 per cent., was abnormal to ordinary physical examination, and 1 was obviously an advanced case of silicosis. Of the remaining 158 men, only 20, or 13 per cent., showed any signs of pulmonary abnormality, a percentage which is probably about that of a general working class male population.

The composition of the local millstone grit and of the materials used at the two ganister sand brickworks shows that while the silica content varies but little (ganister sand 86 per cent. silica, millstone grit 96 per cent.) the clayey content of the ganister sand is at least nine times that of millstone grit. It may well be much greater, as the figure 1.30 per cent., given by a well-known gritstone firm, includes iron and alumina. Thus:

Contents of Sample	*Brick Factory A*	*Brick Factory B*	*Gritstone Company*
	%	%	%
Silica	88.90	84.90	96.40
Alumina	7.42	9.88	1.30
Ferric oxide..	0.16	0.19	
Manganese, magnesium, and tin oxide..	0.44	0.85	0.00
Soda	1.49	0.84	0.00
Lime	0.00	0.34	0.36
Water and loss ...	2.49	3.30	1.94

An analysis made of a Derbyshire silica brick in the usual way gives: silica, 92.79 per cent.; alumina, 4.93 per cent. Rational analysis gives: silica, 82.89 per cent.; clay, 8.58 per cent.; feldspathic matter, 7.93 per cent.

The bulk of the workers in the ganister sand brickworks live in the Bakewell registration district, as do the greater part of the Derbyshire gritstone cutters. An accurate comparison of the phthisis death rates in the two local industries can, therefore, be readily made.

The average numbers resident in the district during the decade 1911 to 1921 may be taken at approximately 160 brickmakers and 350 gritstone workers. A search through the death registers for that period shows that 19 gritstone workers died of phthisis, 8 of pulmonary fibrosis, and 1 of hemoptysis, giving an annual death

rate per thousand of 5.43 for phthisis alone, and 7.71 for phthisis and pulmonary fibrosis taken together. The average age at death was 49.5 years for phthisis and 56.1 years for pulmonary fibrosis.

During the same period only one death from phthisis was registered as having occurred in a silica brickmaker (a man aged 30), giving an annual phthisis death rate of 0.6 per thousand in that industry. The average annual

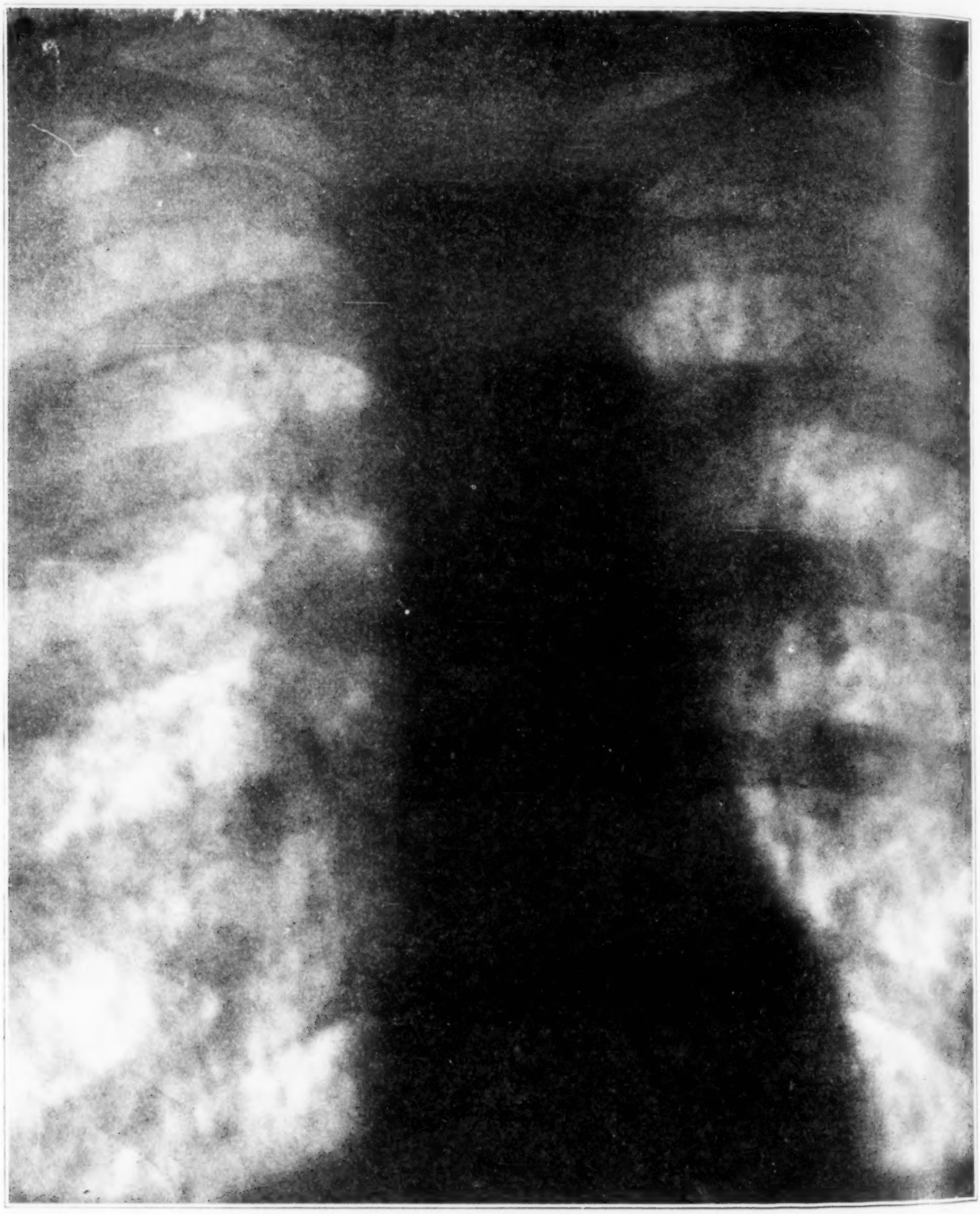

FIG. 1.—J. M., aged 45, a Derbyshire stone cutter for fifteen years. Showing typical "snowstorm" silicotic markings.

phthisis (pulmonary tuberculosis) death rate for the county of Derbyshire for the same decade was 0.71 per thousand. (See Table 1.)

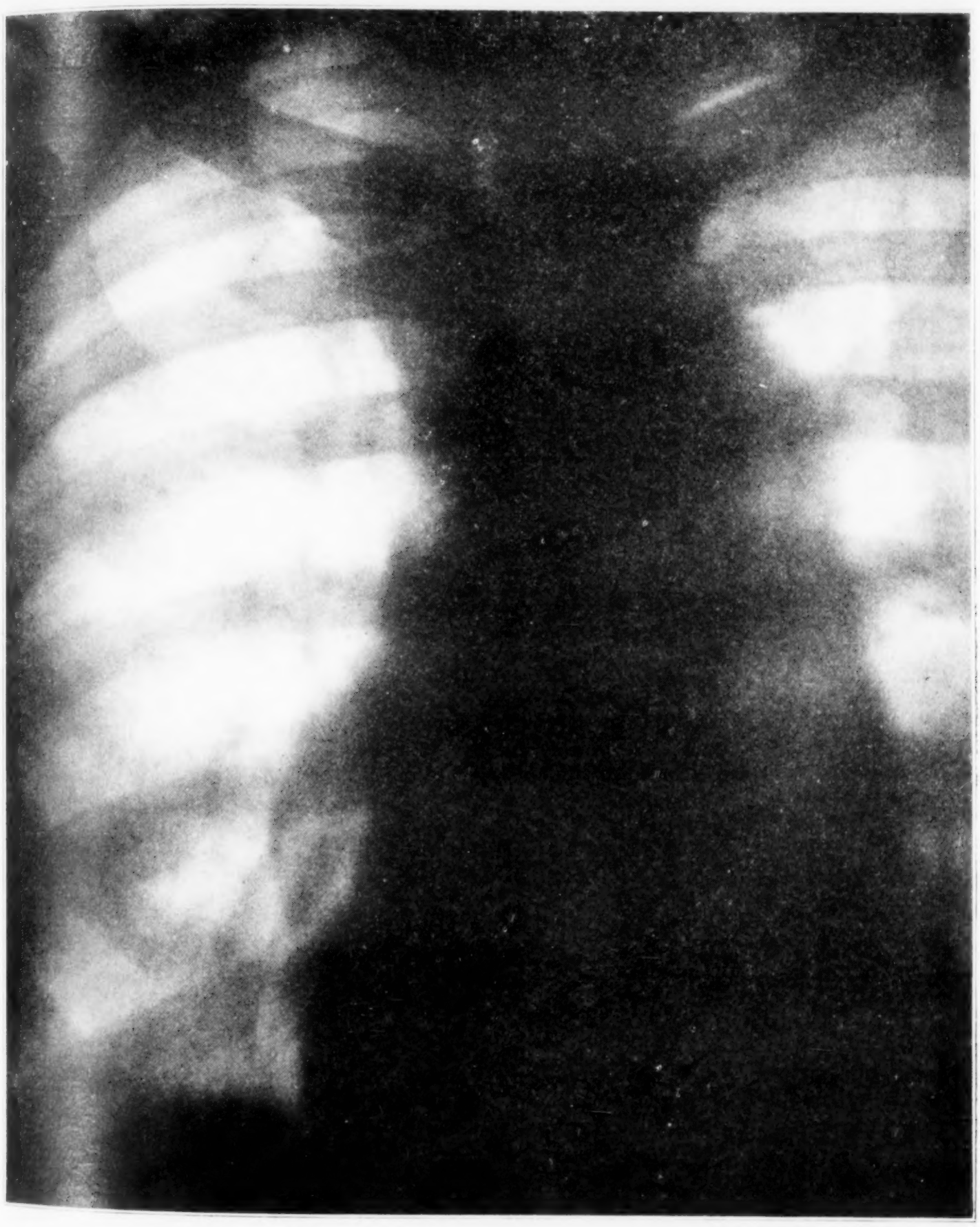

FIG. 2.—J. D., aged 52, a Derbyshire silica brickmaker for twenty-nine years. Showing shadow of a thoracic aneurysm.

It may be of some interest to publish two radiographs, one of a gritstone mason, the other of a silica brickmaker. The former, aged 45, had worked at stone cutting for fifteen years; the latter, aged 52, was a silica brickmaker for twenty-nine years. Both complained of severe dyspnea,

cough, and chest pain on exertion; in both cases the sputum was free from tubercle bacilli; and both were

TABLE 1.—COMPARATIVE DEATH RATES FOR PHTHISIS AND PULMONARY FIBROSIS, DERBYSHIRE, 1911 TO 1921

GROUP	APPROXIMATE NUMBER RESIDENT	DEATHS FROM		ANNUAL MORTALITY PER THOUSAND LIVING	
		Phthisis	Pulmonary Fibrosis	Phthisis	Phthisis and Pulmonary Fibrosis
Gritstone masons	350	19	8	5.43	7.71
Ganister sand brickmakers	160	1	0	0.6	0.6
People of Derbyshire	584, 703	...	...	0.71	....

suspected of silicosis. Radiographic examination showed typical "snow-storm" silicotic markings in the case of the gritstone mason (Fig. 1). The silica brickmaker's lungs were clear, but the shadow of a thoracic aneurysm was apparent (Fig. 2).

The gritstone mason changed his occupation, is alive and working, but dyspneic, 1926; the silica brickmaker died of his aneurysm within eighteen months.

Discussion and Conclusions

The assumption that the comparative absence of pulmonary silicosis in the brickmakers was due to the physical character of the silica sand seemed, at first sight, reasonable.

Later workers, however, have brought forward serious objections to the old mechanical theory of silicosis. Thus Gye and Kettle (6), writing in 1922, state "Hardness, heaviness and sharpness of points or edges are properties which lose their significance when applied to particles of dust enclosed in the cytoplasm of wandering cells; direct mechanical injury to the lung (in the circumstances under which dust gains access) is almost inconceivable." Kettle has further shown that fibrosis results when the tissues of the body are subjected to the influence of soluble colloidal silica.

Finally, there is reason to believe that the relative immunity of the Derbyshire silica brickmakers from silicosis is really due, not to the shape of the silica particles, but to the fact that the material used is not pure silica, but a mixture of about 84 per cent. silica and 16 per cent. clay and earth with some organic matter. It would appear that the presence of these latter materials in some way inhibits the action of the silica dust.

In support of this contention a conclusion in an inquiry by Smith and Collis (5) is to the point:

> . . . . this inquiry has revealed an unsuspected fact, viz.:—that the influence of silica dust in favouring tuberculous infection is modified when the silica is mixed with certain clays. Ganister or other stones

containing a high percentage of free silica are mixed with clay sufficient for binding purposes in the manufacture of certain refractory bricks and other articles; and stone is added in varying proportions to fireclay to increase its refractory properties, or to modify or reduce the amount of shrinkage when subjected to high temperatures. During this inquiry no history has been obtained, up to the present, either from workers or occupiers that a high mortality from phthisis is experienced by those employed in the manufacture of such articles containing clay as an admixture.

The conclusion here quoted is based on the absence of any excess of phthisis mortality among men making ganister bricks in Stirlingshire and at Elland, Yorkshire; the silica used in those places is not fine smooth sand as in Derbyshire, but is spicular, resulting from the fracture of ganister, which is a quartzite rock. One factor, however, is common to the materials used, as instanced above and in Derbyshire, namely, that the silica particles occur in conjunction with clay containing organic matter.

There seems to be little doubt that the relative immunity from silicosis enjoyed by these three groups of silica brick workers is directly attributable to the fact that clay and loam are found mixed naturally with the ganister in the one case, the silica sand of Derbyshire, or are added to the silica used, as in the ganister brickworks of Stirling and Elland, to form the raw material. Exactly how this protection comes about is at present an unsolved but very interesting problem, the solution of which may completely alter our present ideas regarding the prevention of silicosis.

Summary

1. Stonecutting is as old as the human race. In early historic times no references to stonemasons' disease are made, possibly owing to the use of stones, such as limestone, which are not composed of silica.

2. Stonemasons' disease has long been known in Derbyshire where millstone grit, a hard sandstone, is worked.

3. Exposure to fine silica dust is the origin of this disease, which is identical with silicosis as seen in other occupations.

4. Certain makers of ganister bricks in Derbyshire, although exposed to inhalation of fine dust containing about 84 per cent. of silica, do not develop silicosis or experience a high mortality from phthisis.

5. The silica dust arising in the manufacture of ganister bricks contains about 16 per cent. of clay and earth with some organic matter. Apparently this admixture in some way interferes with the usual harmful influence of silica.

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EXPERIMENTAL SILICOSIS*

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THERE is general agreement that the prolonged inhalation of dusts containing silica is dangerous to life: first, because of the chronic fibrotic changes brought about in the lungs by the inhaled dust, and, secondly, because of the abnormal susceptibility to pulmonary phthisis which is apparent in silicotic individuals. But while the facts are accepted, there is by no means complete agreement as to how silica produces its characteristic lesions, or how it aids the growth of the tubercle bacillus in the lungs. The matter is one of considerable difficulty. It is, in the first place, far from easy to reproduce experimentally the conditions to which the industrial worker in silica is exposed. The ordinary laboratory animals are unsuitable for the more obvious inhalation experiments; if it were possible to face the expense of replacing them by monkeys, however, interesting results might be obtained. Short of this, indirect experiments have to be devised, and due allowance in their interpretation must be paid to their artificiality. Among the other practical difficulties with which one is faced in devising experiments is the fact, which is apparently beyond dispute, that certain dusts, though they may contain a considerable proportion of silica, are relatively harmless; while others, though containing smaller quantities of silica, produce the characteristic results in those exposed to them. The problem in itself is not so simple as it at first appears, and no doubt many of the minor clinical discrepancies may necessitate prolonged and rather dull investigation before they are thoroughly understood.

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MODE OF ACTION OF SILICA

On Tissues

The obvious starting point in these investigations is the study of the action of silica, in the pure state, on the tissues. When Dr. W. E. Gye and I started our investigations on silicosis some seven years ago, the generally accepted theory was that quartz or silica produces its effects in the lungs by physical agencies. It was supposed that the process was traumatic, due to direct injury of the cells by the sharp quartz crystals. Even at this time doubts had been cast on this explanation (Collis (1)), and a little thought makes it obvious that it was exceedingly improbable. Such physical attributes as hardness and sharpness can have little significance for particles of the order of size of the silica dust which is inhaled. Moreover, the supposed insolubility of silica is more apparent than real, for it is fairly readily soluble in alkalies.

Direct experiment brought forward evidence that the action of the dust could be explained on chemical lines. If soluble silica, colloidal silicic acid, is injected into the subcutaneous tissues of animals, especially into mice, whose subcutaneous tissues lend themselves particularly well to such observations, a highly characteristic tissue reaction is brought about. There occurs a central mass of necrosis surrounded by a zone of leukocytes; outside this is a zone of spreading secondary necrosis, which is bounded by an area of organizing inflammatory fibrosis. In the course of time the necrotic tissue becomes absorbed and replaced by the invading fibrous tissue, and late lesions are represented by nothing more than a fibrous scar. If instead of using colloidal silica we use insoluble amorphous silica oxide, the same lesion is produced, though it takes rather longer to form. None of the insoluble silica can be recognized in the lesion; it all goes into solution and there can be no reasonable doubt that it brings about its results by its chemical action on the tissues and cells. This becomes strikingly obvious when we compare the local silica oxide lesion with that produced by aluminium oxide, oxide of iron, or carbon, substances which are actually insoluble and remain permanently quiescent and recognizable in the tissues, with only a moderate degree of reaction around them. If now we inject equivalent quantities of mine dust and compare its action with silica, we find that exactly similar lesions are produced. There is in fact an exact parallel between the lesions of insoluble silica oxide and those of mine dust, and it is impossible to escape the conclusion that these two substances become soluble in the tissue fluids and are thus enabled to exert their chemical action.

The final proof of this is, I think, established beyond doubt by some experiments which I have recently been carrying out. Collodion sacs, having an appropriate degree of permeability, are filled with insoluble silica oxide suspended in distilled water and are embedded in the loose connective tissue of the flanks of rabbits. In each animal a similar sac, filled with a solution of calcium chloride, is embedded as a control in the opposite flank. It is found after an interval of time that both sacs become surrounded by an inflammatory reaction, which is much more severe in the neighborhood of the silica sac than in that of the calcium sac. Microscopically the reaction around the silica sac reproduces the picture with which we are familiar in the ordinary subcutaneous silica lesion. These experiments are still in progress and will be published in full detail later. But even as far as they have gone, they show that insoluble silica enclosed in a semi-permeable membrane and exposed to tissue lymph becomes soluble, and passes through the membrane.

We come to the conclusion, then, that the action of silica is a chemical one. Insoluble silica or mine dust becomes hydrolized in the tissues and, as a soluble protoplasmic poison, is able to exert its action on the cells. The fibrosis in local subcutaneous lesions is merely the ordinary healing fibrosis which follows any tissue injury in the usual course of events. Whether the characteristic pulmonary

silicotic fibrosis is produced in this way is not absolutely certain. There is a good deal of evidence to show that it is, but one must not forget the experiments of Gye and Purdy (2), which suggest that chronic poisoning of the reticulo-endothelial cells may stimulate them to the production of collagen without actual previous necrosis.

On Growth of Tubercle Bacillus

To turn to the second part of our problem: How does silica aid the development of the tubercle bacillus in the body? The increased susceptibility of silicotic individuals to tuberculosis has been ascribed to the massive fibrosis of the lung produced by the silica, with its attendant interference with lymph drainage. Much the same idea is expressed by Haldane (3), who says "we can easily imagine how a lung that becomes more and more clogged with dust becomes more and more susceptible to the attacks of the tubercle bacillus." But this does not carry us very far. After all, the lung of the coal miner is clogged and its lymphatic system seriously deranged, but he shows no increased susceptibility to tuberculosis. Even if we accept the importance of the anatomic factor, the essential explanation of the problem is still to be sought. If we are to discover the truth, it is necessary to leave speculation and to put the matter to the test of experiment.

What actually happens if silica is inoculated into animals along with tubercle bacilli? Gye and I (4) in our original experiments found that, if the substances are inoculated in combination into the subcutaneous tissues of mice, animals having very little susceptibility to tuberculosis, an enormously greater proliferation of the tubercle bacillus occurs than in control mice. Studying the process by the technic of "early stages," we found that the injected bacilli proliferate abundantly in the coagulum of the silica lesion, being protected from the cellular defenses of the body. In the course of time the coagulum is absorbed and the lesion becomes infiltrated with phagocytes, which absorb the bacilli. But in the meantime a minimum dose of bacilli becomes a dose of considerable magnitude, and this in itself is of great importance in the determination of an infection.

But why do the bacilli proliferate in the necrotic center of the silica lesion? It may be due to several causes: (1) to the mere mechanical protection of the bacilli during the early days of their lodgment in the body; (2) to the rich pabulum furnished by the disintegrated cells; (3) to the stimulating action of silica on the growth of the bacilli. In the first two possibilities the silica would act simply by bringing about tissue destruction, but in the third its action would be more specific, on the bacilli rather than on the tissues. In order to elucidate this point, I adopted the technic of the fixation abscess (5). In this method bilateral abscesses were produced in the flanks of mice and rabbits, one abscess being produced by silica, the other by some other agent, such as turpentine, calcium chloride, or various other substances. The animals were then inoculated intravenously with tubercle bacilli

and at a later date were killed, and the abscesses excised and examined histologically. It was found that on the whole a far greater determination of the bacilli to the silica abscess occurred than to the abscesses produced by any of the other agents, though, if anything, the degree of necrosis induced by the silica was less. It becomes apparent then that not the mere necrosis of tissue but the specific necrosis or cell damage produced by silica is the factor. I therefore attempted to see if I could modify the development of an artificial miliary tuberculosis in animals by previous treatment with silica.

Rabbits and mice were inoculated intravenously with silica oxide; later they received intravenous doses of tubercle bacilli, and I hoped that, inasmuch as the same type of cell is concerned in the phagocytosis of both silica and tubercle bacilli, miliary tuberculosis would develop more rapidly and more severely in the silica animals than in the controls. In many of my experiments this was so; but in others the results were much less conclusive. And this might have been expected, for in attempting to modify experimentally the reactions of the tissues to pathogenic organisms, it is scarcely possible to obtain absolutely clear-cut results; the changes brought about can differ only in degree. If the resistance of the tissues and the pathogenicity of the organism are as nicely balanced as they are when the mouse is the host and the tubercle bacillus the infecting organism, no agent can bring about permanent modification of the infection, unless its influence is exercised more or less continuously. In the guinea-pig, human tubercle bacilli call forth progressive reactions, and the agent alters the process only appreciably in the early stages; later the ordinary progressive tuberculosis entirely obscures the process. In rabbits, human tubercle bacilli produce retrogressive lesions and, again, it is obvious that the time factor must be of paramount importance, for eventually the reparative process will be predominant in both the modified animals and the controls. Hence the necessity of working on a large scale if conclusive results are to be obtained. Each experiment must contain so many animals that enough may be available for examination at graduated intervals of time to make it certain that any variations observed are really significant and are not merely due to chance.

The third possibility, that silica has a stimulating action on the growth of the bacillus, might be investigated *in vitro*, and I have made several attempts to improve the growth of tubercle bacilli in culture media by adding to these varying quantities of silica. But here again the experimental difficulties are considerable, and my results have been inconclusive.

There is yet another possibility. It may be that the combination of silica and tissue protein results in the formation of an accessory factor which aids the infective process; experiments to test this are now in progress, but it is as yet too early to speak of results. As far as my work has gone, it leads me to this conception of miners' phthisis: Silica inhaled into the lung is picked up by the large phagocytes. Some of these find their way to the lymph nodes at the root of the lung

and there deposit the dust. Others again are excreted on the surface, while still others are arrested in the lymphatics of the lung itself. This arrest tends to occur the more readily since silica is a cell poison, and, if it passes into solution within the bodies of the phagocytes, will mobilize and kill them. Not because of the functional failure of the phagocytes, as Haldane suggests, but because of their death does silica tend to accumulate within the lung, causing fibrosis by one of the methods I have suggested. On the arrival of the tubercle bacillus into a medium rich in silica, whether the bacillus comes from outside, or whether it is already present in a partly healed lesion, it will find itself in peculiarly favorable circumstances for its growth and development.

The silicotic lung appears to be a more favorable medium for the growth of the tubercle bacillus than the normal lung, not because it is fibrotic, nor because its lymphatic drainage is interfered with, but because it contains silica.

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A SHORT DESCRIPTION OF A DUST TRAP TO BE USED IN CONJUNCTION WITH PERCUSSIVE ROCK DRILLS*

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THE apparatus shown in the accompanying sketch (Fig. 1) is designed to trap the dust, produced during rock drilling, as it leaves the hole, and to deposit it into a container from which it can be emptied as desired.

The following is a brief description of the principle of the apparatus, reference being made from time to time to the figure, from which the body of the drill is omitted.

A rubber hood, 1, is placed over the drill hole. This hood is provided with two holes, one through which the drill steel, 2, passes, and the second to which is attached a short length of flexible steel tube, 3.

The flexible steel tube, 3, is attached to a rigid steel tube, 4, on the end of which is fixed an ejector, 5. To the end of the ejector is attached a length of 2-inch indiarubber tube, 6, leading to the dust filter which consists of a flannel bag, 7, to the lower end of which is attached a container, 8.

The ejector, 5, is operated by exhaust air led from the drilling machine by the rubber tube, 9. This ejector creates a suction in the rubber hood, 1, and consequently, as dust is produced it is carried along the tubes 3 and 4, through the ejector, from which it is exhausted into the dust filter. Here the dust, caught on the flannel of the filter bag, is shaken loose by successive puffs of air escaping from the nozzle of the ejector and settles into the steel container, 8, while the air filters through the flannel bag, 7, and escapes into the atmosphere almost entirely free of dust. The principle of using the exhaust air from the drill insures that the induced draft is brought into play whenever the drill is in action, and that the more rapidly the drill works the greater is the draft induced by the greater amount of air escaping. The steel container is of sufficient capacity to hold the dust produced by about twenty 5-foot holes, and it is usually emptied at the end of each shift.

The whole device can be supported by a vertical stand as shown in the sketch; an alternative method of support, often useful underground, is to have a horizontal adjustable strut jacked between the walls of the drift. The tube, 4, passes through a sliding bracket, fitted with a swivel and

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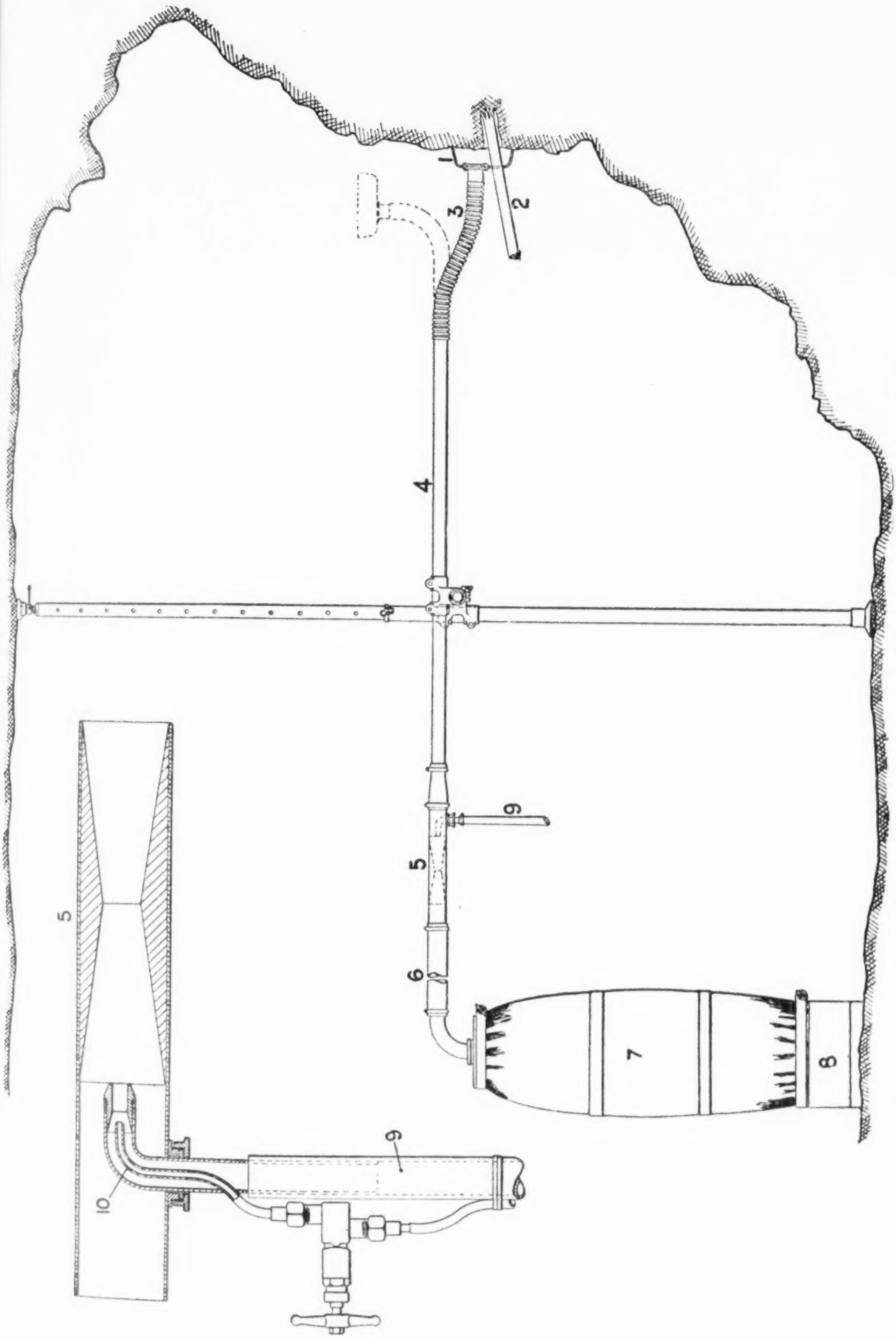

FIG. 1.—Dust trap for use in conjunction with percussive rock drills.